LIBRARY
RECEIVED
FEB 7 1911
U. S. Department of Agriculture

Price One Shilling Net,
or 25 Cents.

February, 1911.

THE VETERINARY JOURNAL

A Monthly Review of Veterinary Science.

Editors for Great Britain and Ireland:—

FREDERICK HOBDAY, F.R.C.V.S., F.R.S.E.,
FORMERLY PROFESSOR IN THE ROYAL VETERINARY COLLEGE, LONDON.

GEO. H. WOOLDRIDGE, F.R.C.V.S., M.R.I.A.,
PROFESSOR OF HYGIENE, MATERIA MEDICA AND THERAPEUTICS IN THE ROYAL VETERINARY COLLEGE, LONDON.

Editor for Australasia:—

J. A. GILRUTH, D.V.Sc., M.R.C.V.S., F.R.S.E.,
DEAN OF THE VETERINARY FACULTY, UNIVERSITY OF MELBOURNE, AUSTRALIA.

Annual Subscription for the British Empire, 12s. Post Free; for the United States, $3.00.

LONDON.
BAILLIÈRE, TINDALL & COX,
8, HENRIETTA STREET, COVENT GARDEN.

AUSTRALIA AND NEW ZEALAND.
Messrs. GORDON & GOTCH, Ltd.,
Melbourne, Sydney, Wellington, &c.

NEW YORK:
W. R. JENKINS,
851 and 853, Sixth Avenue.

CHICAGO:
ALEXANDER EGER,
42, La Salle Street.

Entered as Second-Class Matter in the U.S. Post.

No. 428. VOL. 67.

Instruments

For the New Treatment of

ROARING . . .

As made for and used by Professor HOBDAY and other leading Members of the Profession.

Vide "VETERINARY JOURNAL," January, 1911.

Retractor.

Special Knife.

Operating Forceps.

Swabbing Forceps.

Prices on application to—

ARNOLD & SONS,

Veterinary Instrument Manufacturers,

6, Giltspur Street, West Smithfield, London, E.C.

Telegraphic Address:
"INSTRUMENTS, LONDON."

Telephone Numbers:
CITY 5240 (3 lines).
HOLBORN 518.

CONTENTS.

	PAGE
EDITORIALS—	
Intertransmissible Human and Animal Trypanosomes	65
A Clean Milk Supply...	66
GENERAL ARTICLES—	
Remarks on Milk Fever from a Clinical Aspect. By E. WALLIS HOARE, F.R.C.V.S.	67
Gangrenous Foot-diseases of Cattle (Bacterial Necrosis). By H. A. REID, F.R.C.V.S., D.V.H.	72
Facial Dermatitis (Facial Eczema) in Sheep in New Zealand. By H. A. REID, F.R.C.V.S., D.V.H.	78
The New Operation for Roaring—A Question in Historical Accuracy. By W. L. WILLIAMS	80
Experiments to Ascertain if Cattle may act as a Reservoir of the Virus of Sleeping Sickness (*Trypanosoma Gambiense*). By Colonel Sir DAVID BRUCE, C.B., F.R.S., Captains A. E. HAMERTON, D.S.O., and H. R. BATEMAN, R.A.M.C.; and Captain F. P. MACKIE, I.M.S.	87
Rabies and its Control in India. By Captain E. CLIVE WEBB, F.R.C.V.S. ...	93
CLINICAL ARTICLES...	105
CANINE CLINICAL NOTE—	
The Treatment of Eczema by Intramuscular Injections of a Solution of Collargol. By J. A. N. DA CUNHA, G.B.V.C.	114
ABSTRACT AND REPORT—	
A Method for Isolating and Growing the Lepra Bacillus of Man and the Bacillus of Johne's Disease in Cattle (Preliminary Note). By F. W. TWORT, M.D.	118
REVIEWS	123
TRANSLATIONS	125
LETTERS AND COMMUNICATIONS, &c.	128
BOOKS AND PERIODICALS, &c., RECEIVED	128

"SANITAS"

Fluids, Powders,
Soaps, . . .
Dog Soap, . .
Sawdust, . .
Vet. Ointment, .
Embrocation, &c.

Best Disinfectants

For Kennel, Farm and Stable use.

"Sanitas" Sawdust

Is now on Sale in 1/- Bags at all Stores and Chemists. . . . .

Small Samples on application.

THE "SANITAS" Co. Ltd.
LIMEHOUSE, LONDON, E.

TRADE MARK.

SUPPLIED ONLY TO THE PROFESSION.

TUBERCULIN and MALLEIN.

In Hermetically-sealed Ampoules, containing about 4 cc., 9d. each.

Veterinary Tetanus Antitoxin ... ... 10 cc. 2/6; Dry form 3/-
Anti-Streptococcus Polyvalent Serum ... 10 cc. 2/6; Dry form 3/-
Canine Distemper ... ... 4 cc. 2/6

PASTEUR VACCINE CO.'S TUBERCULIN for Diagnosis of Tuberculosis,
PASTEUR VACCINE CO.'S MALLEIN for Diagnosis of Glanders,
ARE INSISTED UPON BY
Foreign and Colonial Governments importing BRITISH STOCK.

"BLACKLEGINE."

CORD FORM OF BLACKLEG VACCINE.

Single, 10 doses, 6/-; 20, 10/-; 50, 24/-.
Double, 10 doses, 8/-. Needle Outfit, 2/6; Ditto with Forceps, 3/-.

200,000 doses have been sold in the United Kingdom. The Easiest Method of Inoculation, and always ready for use.
We also furnish Anthrax and Blackleg Vaccine (powder form); Rat Virus, White Scour Serum, &c.

VERGOTININE

Pasteur Vaccine Co.

FOR BROKEN WINDED HORSES.

And Bronchitis Glandon. Persistent Cough, Chronic Catarrh, &c.

Prepared by A. VELPRY, Billancourt, Boulogne, S. Seine, France.

Cure guaranteed in one month. Thousands of recommendations. Write for full particulars.
Specially Reduced Price to Veterinary Surgeons, 6s.

PASTEUR VACCINE COMPANY, LTD., LONDON, PARIS, &c.
C. H. HUISH & CO., Surgical Instrument Makers,
Telegrams—PROSUM, LONDON. Telephone—4639 Central.
General Agents for Great Britain and Ireland,
12, RED LION SQUARE, LONDON, W.C.

Antiphymatol Protective and Cure Vaccine Matter against Cattle Tuberculosis.

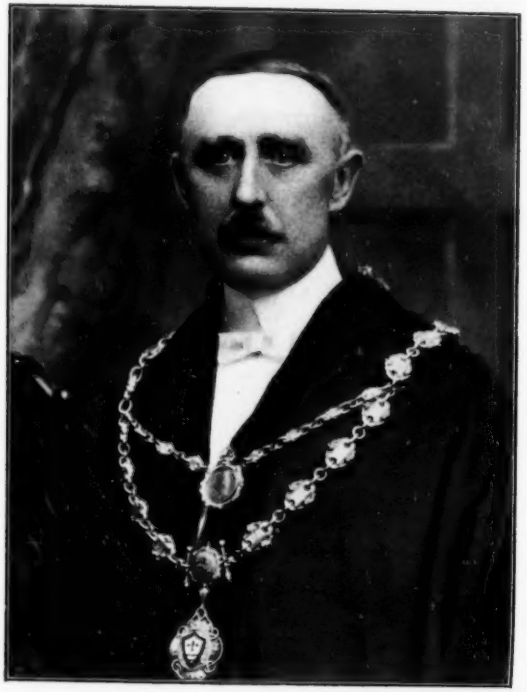
Phymatin for the Ophthalmoreaction for the Perception of Cattle Tuberculosis.

Malleïn for Injection ready for use, and for the Ophthalmoreaction.

Mice-Typhus-Bacillus, for the certain destruction of Fiel and House Mice.

HUMANN & TEISLER, Dohna, b. Dresden.

MR. R. C. TRIGGER, J.P., F.R.C.V.S.
Mayor of Newcastle-under-Lyme.



MR. R. E. L. PENHALE, J.P., M.R.C.V.S.
Mayor of Torrington.

(*For Biographical Note see* VETERINARY JOURNAL, *January*, 1911.)

MR. R. E. L. PENHALE, J.P., M.R.C.V.S.
Mayor of Torrington.
(See Biographical Note in Veterinary Journal, January, 1911.)

THE

VETERINARY JOURNAL

FEBRUARY, 1911.

Editorials.

INTERTRANSMISSIBLE HUMAN AND ANIMAL TRYPANOSOMES.

A Correction and an Explanation.

Indexed B. A. L.

In this issue of the Veterinary Journal is to be found an article from the *Proceedings of the Royal Society*, by Colonel Bruce and others, pointing out that cattle in infected zones may act as a reservoir of the virus of sleeping sickness. We made reference to this in an editorial note in our issue for January, and followed it with some comments on a communication which we had received for publication from Mr. Bevan. This article appeared on pp. 41-47 of that number, and concerned a human trypanosome obtained from an European who had apparently contracted the disease in Northern Rhodesia. Bevan had successfully inoculated this trypanosome into the following species —viz., rabbit, guinea-pig, rat, mule, and sheep—but at the time of forwarding us the note he had not been able to definitely identify it. In this connection Bevan said, "It has been suggested that the human trypanosome of Northern Rhodesia may not be *Trypanosoma gambiense*, but some animal trypanosome habituated by the method of transmission or passage to the human host."

A few days before receiving Mr. Bevan's article by post, we received through the courtesy of the Director of the Sleeping Sickness Bureau a copy of the following cablegram, sent through the British South Africa Company: "Communicate the following to Sleeping Sickness Bureau from Bevan. Referring to report posted December 3, cattle trypanosome from North Eastern Rhodesia now recognised as *T. vivax*. Please correct and inform Wooldridge. Veternosis." On receiving the article we published last month, we naturally supposed the cablegram to refer to it, and we accordingly

inserted a footnote embodying the purport of the cablegram. This was an unfortunate error, and we are grateful to the Director of the Sleeping Sickness Bureau for drawing our attention to it, and pointing out that the cablegram had reference to an article published by the *Veterinary News* of January 7, entitled, "Note on a Trypanosome found in Cattle from North Eastern Rhodesia." Evidently the copy of the cablegram should have gone to the Editor of the *Veterinary News*, and not to us.

Commenting on the matter last month, we said: "This trypanosome (*T. vivax*) is known as causing disease in cattle, sheep, and goats in the Cameroons, but we are unaware of it having been previously found affecting human beings." Our hesitancy was completely justified, and we hasten to correct the unfortunate error.

In a footnote to a summary of Mr. Bevan's paper in the *Bulletin of the Sleeping Sickness Bureau*, the Editor points out that the human trypanosome referred to is that described by Stephens and Fantham as *T. rhodesiense*, and is quite distinct from *T. vivax*.

W.

A CLEAN MILK SUPPLY.

We have received three leaflets prepared by the Joint Committee on Milk of the National Health Society and the National League for Physical Education and Improvement, dealing with the above subject. They consist of instructions to three different classes of people associated with milk, viz., the producers, the distributors, and the consumers respectively, and their object is to assist in obtaining a clean and wholesome milk supply, or at least to reducing the various chances of contamination and ill-effects as far as possible under present conditions. If the persons addressed in the respective leaflets will follow the advice offered, they will do so with practically no expense to themselves, and will do much to achieve the very desirable objects for which the above-named societies have been brought into existence. We reproduce on pp. 116-118 in full a copy of Leaflet A, which is perhaps of the highest importance. As will be seen, it is written in clear and lucid language, free from technicalities, and in such a way as to be easily understood by anybody who can read. The other leaflets are equally clear and concise, and may be purchased very cheaply. Any person interested in the subject would do well to buy some and distribute them for the education of those who are not well informed in these matters.

General Articles.

REMARKS ON MILK FEVER FROM A CLINICAL ASPECT.

By E. WALLIS HOARE, F.R.C.V.S.

Cork.

Indexed B. A. L.

It is only those having experience of country practice who can really appreciate the great boon conferred on the owners of cattle by the introduction of the Schmidt treatment of milk fever. Previous to this the mortality from this disease was so high that every form of treatment was in turn tried, but found to fail.

Probably had the practitioner, who discovered the successful treatment, devoted his attentions instead to speculations as to the exact cause of the disease, or discovered a bacillus without finding a measure to combat its effects, he would long ere now be accorded a high place in the scientific world, and be the recipient of many honours.

However this may be, and no matter how many theories may be advanced to explain the etiology of the disease, we have at last a reliable form of treatment, although we cannot explain the *modus operandi* of the therapeutical measure adopted. Empirical, no doubt, the treatment is, but, nevertheless, it proves eminently successful. But like all other diseases, a variable number of fatal cases occur in spite of treatment. Some practitioners, indeed, claim that under the present system of treatment they never lose a case.

This may be attributed to the fact that treatment is resorted to on the appearance of the primary symptoms of the disease; or the practitioners may belong to that fortunate class of lucky individuals who "never lose a colt after castration," and they have similar results with milk fever. As, however, I do not happen to belong to that class, I find that a certain number of cases of milk fever die in spite of treatment. The cause of death in many of these cases is difficult to discover, but in some at least it is possible to suggest an etiological factor.

From a clinical aspect these cases may be referred to certain groups, and in passing I may remark that it is not the patients who show the most severe cerebral symptoms at first that supply the largest number of fatal cases.

There is no doubt but that the earlier professional attendance is availed of, the fewer will be the number of fatalities; hence the in-

telligence of the owners is an important factor in the question of results.

Group A.—Those in which the owner has administered the ubiquitous dose of Epsom salts before the practitioner arrives.

Fortunately these cases are becoming less frequent in the present day, as the owners are commencing to appreciate the fact that drenching in any shape or form is not only useless, but dangerous, and frequently produces fatal results in this disease.

In consequence, however, of the inherent love for drugs possessed by mankind, it is often difficult to persuade some owners of animals that any drugs administered by the mouth cannot produce any beneficial effects in milk fever, and are far more likely to do harm. I have always observed that the effect of Epsom salts in this disease was to add to the depression already existing, while in a large number of instances inhalation broncho-pneumonia was produced, which only showed marked symptoms after the animal had regained consciousness.

Group B.—Cases in which consciousness returns after treatment, but there is complete loss of power in the hind limbs.

These furnish a large proportion of the fatal cases, as in spite of every form of treatment the paralysis remains, and the animals are unable to rise.

In some instances the hypodermic injection of full doses of strychnine, and the application of strong counter-irritants to the spine may produce desired results, but in my experience recovery is the exception. What the lesion is in the spinal cord that is responsible for the paralysis I have not the slightest idea.

No one now doubts that milk fever may appear *prior* to parturition, and in my experience such cases often terminate in persistent paralysis.

Some authors describe a form of paralysis occurring just prior to parturition, and another—*post-partum* paralysis—appearing after parturition, but I fail to see how these are to be differentiated from cases of milk fever.

Group C.—Cases that die in rather a sudden manner, without any appreciable cause.

These are not common, and the sudden occurrence of death would suggest syncope. The animals regain consciousness in the ordinary manner, get on their feet, commence to feed and ruminate, and, after a variable period, die suddenly; in some instances hæmorrhage from the nostrils has been observed.

Group D.—Cases in which relapses occur. The animal apparently recovers in the ordinary manner, but next day is found down again, and all the symptoms return. On again distending the udder with air, she may regain consciousness, get on her feet, and in a variable period go down a third time, never to rise again, as a rule. These cases are very annoying and difficult to account for. In my experience it is not the cows in specially high condition that furnish most of these relapsing cases, indeed many of them are in poor condition.

I have observed that if the udder be in a flabby condition, containing little milk, and not inflating easily when air is pumped in, the case is likely to do badly. Of course, this may only be a coincidence, but I have observed it on several occasions. Cows subjected to chills, exposure on long railway journeys after calving, especially in cold wet weather, are likely to prove bad subjects for the disease.

Cows recently calved, and purchased by dealers at country fairs, are well known to be liable to milk fever, whether in high condition or otherwise.

In this district, during the present month (November), the disease has been very common, and certainly cows at this season are not in high condition, while the weather has also been very cold and wet. I have come to the conclusion that the influence of high condition and hot weather in the production of the disease has been greatly exaggerated. No doubt the *sudden* appearance of hot weather does bring an increase in the number of cases, but what about the large proportion that occur during the winter months?

Can we adopt any precautions to prevent the occurrence of fatal cases? I think there are some little details which seem to turn the scale in favour of the patient.

(1) With reference to milking. No milk should be removed from the udder until the following day, after the "pneumatic" treatment has been carried out. Even then the udder should not be milked dry. I have often observed that it is after complete milking has been carried out that the animal goes down again.

(2) With regard to food, the animal should be kept on spare diet for a few days. A rumen filled up with food seems likely to bring about a relapse, as some cows commence to feed ravenously after regaining consciousness and getting on their feet.

(3) The cow should not be left out on grass too soon, especially when the weather is hot. Hot, ill-ventilated buildings have also a tendency to induce a relapse.

In a word, moderation in milking, in food and in exercise are to be observed.

Clinical observation of a large number of cases of milk fever under various conditions, shows one that different types of the disease are met with. Many intelligent owners now call in professional assistance when the primary symptoms of the disease present themselves.

These symptoms are generally paddling with the hind feet and a staggering gait, sometimes associated with a dazed appearance of the eyes and countenance. These symptoms often come on shortly after milking has been carried out.

The practitioner proceeds to adopt the "pneumatic" treatment, while the cow is standing, but it often occurs that during the process she staggers and goes down. Such cases generally do well.

Mild cases are sometimes met with, so mild, that by the time the practitioner arrives the animal is rapidly improving. Still, the "pneumatic treatment" has to be adopted as a matter of precaution, as well as on the plea that "something must be done."

Cases characterized by marked delirium are common, some of these necessitate the application of restraint to the limbs before the pneumatic treatment can be applied.

The "calmative" effect of the injection of air in such cases is simply wonderful to behold, far exceeding that of chloral hydrate. Cases are met with in which deep coma sets in early, but even here the "pneumatic" treatment exerts remarkable effects. One peculiarity I have observed with reference to milk fever, is that a series of successful cases is the rule in some seasons, while in others, one encounters a run of deaths, fortunately the latter are usually few in number, but still they prove discouraging, and to unreasonable owners they suggest some lack of skill on the part of the practitioner.

With reference to cases that are said to occur some months after parturition a certain amount of doubt may exist, but the effect of the "pneumatic" treatment would lead one to believe that the affection was at any rate similar, if not identical, with milk fever.

It is not unreasonable to suggest that if a case showing certain nervous symptoms recovers rapidly after injecting air into the udder, the affection is milk fever, or a type of that disease. Even shortly after parturition symptoms may appear which are not diagnostic of milk fever, and an element of doubt may exist as to the diagnosis.

There is a tendency to attribute any symptoms shown shortly after parturition to milk fever, and no doubt at times cases of the disease are said to recover, when in reality the symptoms were due to other causes. And I must admit that I have had considerable difficulty at times in coming to a conclusion in certain cases showing

obscure symptoms, such as loss of appetite, accelerated respirations, dulness, inaptitude for exertion, occurring shortly after parturition, as to whether these symptoms were to be attributed to the commencement of milk fever or to some other affection.

Probably when in doubt it is advisable to treat the udder, as otherwise on our departure symptoms of milk fever may develop.

One great advantage of the "pneumatic" treatment which distinguishes it from many others is that it cannot do the patient any harm, even if the disease does not happen to be one of milk fever. This cannot be said for many therapeutical measures.

As regards the treatment of the disease there is little to be said.

The well-known success of the treatment by filling the udder with air, has placed this method above all others.

Simple in administration, perfectly safe, as it can be repeatedly carried out without any risk of mammitis occurring, and, above all, successful.

As regards attempts to sterilize the air, this is entirely unnecessary, although it may look well and assist in "playing to the gallery."

I have treated cases in the most filthy cow-houses imaginable, pigs also inhabiting the building, and the air of a nauseating character, but I omitted attempts at sterilization of the air, and the results were satisfactory.

I shall not attempt an academical discussion on the question as to the therapeutical action of the air, but I have always observed that the more firmly the udder is distended the better the results.

There may be a grain of truth in the mechanical theory of its action. But as we do not know anything definitely with reference to the pathology of the disease, it is difficult to explain how the pneumatic treatment really acts.

One term, however, should be rigidly excluded from the nomenclature of the disease, and that is "apoplexy," as, if the semi-comatose condition depended on this lesion, one fails to see how inflating the udder could cause a disappearance of the symptoms and recovery in a short space of time.

With reference to prevention, many measures have been suggested. It is often said that if the calf be left with the cow for some days the disease will not occur.

Having seen a number of cases occur when this measure was adopted, I doubt its efficacy. I find the most important means of prevention is with reference to the milking. If the cow prior to calving shows a considerable amount of milk, milking should be

carried out. After calving, the cow should not be milked dry for the first few days; the milking should be carried out at intervals.

Probably this is why leaving the calf with the cow may exert a preventive influence, as the milk is gradually withdrawn.

Some time ago the above preventive measure was described as emanating from a Continental practitioner, but in reality it has been known in Ireland for many years, and advised by practitioners.

No doubt it is also of importance to limit the animal's diet for some time prior to parturition, in the case of cows in high condition.

The practice of giving preventive drenches for the disease, however useful from a commercial point of view, cannot be described as rational therapeutics.

For inflation of the udder with air, a very useful air-pump is supplied by Hauptner, Berlin. It is a form of foot-pump, and the required degree of distension of the udder is accomplished with no fatigue to the practitioner, and very quickly. By means of a proper length of tubing, the operator is well out of the way of the animal's hind legs, which is a matter of importance in the case of cows during the exciting stage of the disease. The instrument is portable and simple, and it permits of a higher degree of udder distension than any other I have tried.

A few drops of ol. eucalyptus, or of ol. fœniculi, placed on the wool in the filter portion, exhales a pleasing odour during the working of the instrument, and gives the onlookers the idea that something possessed of miraculous powers issues from the instrument.

For some degree of mystery must enter into the treatment of even a disease like milk fever, as a sort of set-off to the efforts of philanthropic (?) professors (*sic*) who, by writing newspaper articles and lecturing on the subject, endeavour to perpetuate the pernicious system of "everyone his own cattle doctor."

GANGRENOUS FOOT-DISEASES OF CATTLE (BACTERIAL NECROSIS).

Indexed B. A. I.

By H. A. REID, F.R.C.V.S., D.V.H.

Officer in charge of the Veterinary Laboratory, Wellington, N.Z.

Gangrene of the feet, principally affecting cattle, has been frequently reported from various parts of New Zealand. The disease appears to be endemic in certain localities. Reports on the appearance of cases of this nature have been almost exclu-

sively confined to the North Island, a fact which will be considered in relation to the cause of the condition.

In certain districts such outbreaks in the past have nearly always been referred to the effects of ergot. It is possible that in certain instances ergot may share indirectly in the production of the gangrenous lesions, but I consider that it yet remains to be proved whether the ingestion of ergotized food has any special significance as a potential factor in the etiology of the disease.

Shortly after rejoining the Department in 1908 I had an opportunity of investigating an outbreak of gangrene affecting cattle. The trouble had been almost unanimously declared by farmers in the locality to be due to ergot poisoning. The result of my investigations then led me to doubt whether ergot could be held responsible, and later observations have convinced me that true ergotism must be a rarer condition than is generally supposed, since I have never yet observed the classical symptoms attributed to this form of poisoning. My conclusions regarding the nature of the outbreak referred to were embodied in a report to the Chief Veterinarian, from which the following remarks are extracted:—

"None of the cattle on adjacent farms appear to have been attacked, and the owner himself, an intelligent farmer, does not ascribe the trouble to the effects of ergot. Moreover, none of the symptoms generally recorded of poisoning by this agent are present—such, for example, as gastro-enteritis, congestion and hæmorrhages of the visible mucous membranes, emaciation, abortion, and the usual history of occurrence during cold weather. . . . In the absence of more definite information, I do not feel satisfied that the symptoms are the result of ergotism, and have therefore to conclude that they are due to some topical irritant of undetermined origin."

The symptoms which generally characterize the affection are as follows: As a rule, heifers or milking-cows are attacked towards the fall of the year or during winter. A keen observer will notice that certain of the animals appear restless, and disinclined to support weight on one particular leg, generally a hind leg. In the bails cows may be seen to stamp or paw the ground with the affected foot. Later on, lameness becomes apparent, gradually becoming acute, and accompanied by painful swelling of the pastern and fetlock. Constitutional symptoms, in the

shape of loss of appetite and condition, fever, constipation, and diminution of the milk-secretion accompany the appearance of the local lesion. In about two or three days the swelling breaks above the coronet, discharging purulent contents. Gangrene of the affected portion of the limb then sets in; the lesion extends, and involves the tissues below the cannon bone. The claws, or even the whole of the foot, may slough off from the joint, leaving the denuded bone exposed. Above the gangrenous area the limb remains in a normal condition, a sharp line of demarcation, composed of congested and œdematous tissue, existing between the gangrenous and healthy portions. There is complete loss of sensation below this area, but the blood-vessels may still be active, the gangrenous part bleeding on manipulation. It is most common to find only one leg affected. As a general rule, when taken in time and properly treated, the disease runs a benign course. Resolution takes place in about twelve to fourteen days. In consequence of the diminished milk-supply and loss of condition of the affected subjects, owners frequently suffer serious monetary loss, while the dressings and attention which must be given in order to promote recovery occupy a considerable amount of time which might otherwise be more profitably employed.

An Allied Condition Affecting Pigs.

A number of cases presenting similar though differently situated lesions have come under observation affecting pigs. In these animals the snout and skin covering the head and sides of the shoulder are generally the sites of the necrotic lesions. These take the form of a spreading ulceration, giving rise to great irritation and loss of condition. Occasionally the skin and integument around the eyes are involved. The resulting inflammation affects the conjunctiva, inducing a purulent discharge from the eyes, which may lead to temporary or permanent loss of sight. In very severe cases the ulcerated surface may extend over an area from two to three inches up to the size of a dinner-plate in dimension, giving rise to deep excavations, surrounded by a hard, fibrous-looking growth. Under suitable treatment recovery can usually be effected, although in young animals serious mortality may follow with embolic, necrotic lesions in the liver and kidneys.

The essential cause of these conditions in both cattle and swine has been determined to be due to a micro-organism known technically as the *B. necrophorus* (Flugge). This organism—first isolated by Löffler in 1884 from cases of so-called "calf-diphtheria"—has since been observed and studied by various authorities, notably Bang, McFadyean, Schmorl, Mohler, and Morse. Bang, on account of the characteristic lesions following infection, termed it the "necrosis bacillus." The appearance generally assumed by the bacillus is in the form of long, slender filaments, measuring 80 to 100 microns in length, and from 0·75 to 1·5 microns thick. Shorter bacillary forms are also frequently observed, the shortest, in fact, resembling cocci. The organism is thus polymorphic. It is a strict anaerobe, only developing in the absence of air or oxygen. The necrosis organism has evidently a wide distribution in nature, leading a saprophytic existence in manure and in the soil of certain localities. Bang has demonstrated its presence in the intestines of healthy pigs, and according to Nocard and Leclainche it may be regarded as an occasional or habitual habitat of the alimentary canal of all herbivorous animals. Among the smaller animals, rabbits and mice are particularly susceptible to infection. They have been repeatedly employed to recover the organism after inoculation with material derived from a naturally-infected source. Guinea-pigs are, in our experience, under ordinary conditions, immune. The necrosis bacillus is innocuous to man, unless, according to Schmorl, it is associated with the presence of pyogenic organisms, which then render infected wounds very difficult to heal. Doubtless the same remarks would apply in the case of the lower animals. We have found it constantly associated with cocci, apparently the *Staphylococcus citreus*. The *B. necrophorus* gains access to the tissues through the skin, and the factors favouring infection may be classified as mechanical, chemical, and vital. As an instance of the first group, any injury sustained resulting in a wound or abrasion may be taken. Under chemical agents may be placed influences, such as cold, continued exposure to wet or damp, or malnutrition of the skin through digestive disorders, leading to an eczematous eruption and irritation. Vital agents comprise the various bacteria present in the skin, which, under certain conditions, are capable of setting up inflammatory changes deleterious to the histological elements. One or more of these factors may be held responsible for the

entrance of the necrosis organism, which can only exert its peculiar effects when the skin-surface has been injured or has otherwise undergone some abnormal change. The ingestion of ergot may be cited as a possible factor included under chemical agents. It is probable that ergot may induce some constriction of the arterioles, resulting in defective vascular nutrition of the skin. It appears to be well established that necrosis of the feet more frequently occurs in cattle known to have been grazing on ergotized pastures. It is also a fact that the condition occurs on pastures wherein no evidence of the operation of this factor can be traced.

In my experience typical symptoms of ergotism have never been observed; hence in the majority of cases investigated it would not have been possible to conclude that ergot alone was responsible for setting up this condition. I would recommend that experiments be carried out to decide how far the toxic properties of ergot may influence the production of the gangrenous changes.

Introduction of the *B. necrophorus* into the tissues of susceptible animals leads to a necrotic caseous degeneration of the surrounding parts. The centre of such caseous débris is, as a rule, devoid of bacilli. The organism should be sought at the margin of the degenerated and healthy tissue some depth from the surface. Stained preparations, examined microscopically, reveal the bacilli arranged in a filamentous mass, easily mistaken on casual observation for fibrin or loose shreds of areolar connective tissue. The *B. necrophorus* is easily stained by the aniline dyes: Löffler's alkaline methyline blue, or Ziehl's carbol-fuchsin give excellent results. It does not stain by the method of Gram. The study of its physiological effects indicates that the necrosis organism elaborates an active soluble toxin which proves fatal to rabbits in from five to ten days. Generally, prior to death the animal is thrown into tetanic convulsions produced by the action of the absorbed toxin upon the central nervous system.

A significant point in relation to the study of bacterial necrosis is the fact that, so far as observation goes, it appears almost wholly confined to the North Island. No doubt the habits of the *B. necrophorus* are well adapted to the comparatively mild and humid climate of the North Island, permitting it to remain for a longer period in an active condition in the soil or excreta.

From a knowledge acquired by a study of the occurence of the bacillus in nature, one would expect that cattle on old, soiled pastures, or those occupying dirty yards contaminated by an accumulation of fæcal deposits, would be more prone to become affected. This, however, does not prove to be invariably the case. Cattle on freshly-cleared land are sometimes found to be affected. It is noticeable that certain paddocks appear more liable to convey infection than others. These may probably afford in some way a more suitable environment for the propagation of the bacillus.

Treatment.—The treatment of this gangrenous condition calls for the energetic application of remedies which will relieve the pain caused by the inflammatory changes, and, further, destroy the action of the pathogenic organism. In cattle, whenever the first symptoms of tenderness and swelling of the coronet are observed, the part should be thoroughly cleansed with hot water containing a little soda. If practicable, a hot bran poultice, which has been prepared with the addition of a little disinfectant—such as non-poisonous sheep-dip—may be applied, and maintained in position, to promote the softening of the tissues and ultimate suppuration. The poultice must not be allowed to become cold, and should be changed at intervals during the day. By this means acute symptoms are usually relieved, and treatment should now be confined to keeping the affected parts clean, and the application of some reliable antiseptic. Carbolic acid, in the proportion of 1 part to 20 of water, has been found serviceable, or ointments composed of boracic acid or bluestone may be employed. In severe cases, where the sloughing process is already well advanced, the loose, gangrenous pieces of skin or horn should be removed by means of a knife or hot iron, leaving the unhealthy tissue exposed to the air. Antiseptics may then be applied, or the diseased parts may be lightly smeared with Stockholm tar. A saline purgative drench of Epsom salts should, as a rule, be administered, and the bowels regulated by allowing a plentiful supply of grass, or laxative feed if obtainable. On account of the lameness, rendering movement extremely painful, it will often be found necessary to cut and carry the feed to the patient. In all cases it is wise to separate the affected animals from the healthy. Cow-yards and milking-bails must be kept thoroughly clean. Old yards should be dug up (or, preferably,

have their surface soil for at least 6 in. depth entirely removed) and well lined, since the accumulated droppings may contain the *B. necrophorus* in abundance, which on the slightest abrasion or injury may easily gain access to the feet.

The treatment of affected pigs may follow on the above lines with necessary modifications. The ulcerated surface should be cleansed, all dead and diseased tissue being removed by scraping, or the *light* application of a hot iron. The wounds may then be dressed with carbolic-acid solution (1 to 20), or dusted over with a powder composed of calomel (1 part) and boracic acid (10 parts). In the case of pigs, the sties should be cleansed and disinfected, and the surroundings dug up and limed. By the adoption of these principles the trouble may be checked, and recovery brought about. Neglect of treatment too often entails the loss of the affected animals.

FACIAL DERMATITIS (FACIAL ECZEMA) IN SHEEP IN NEW ZEALAND.

Indexed B. A. I.

By H. A. REID, F.R.C.V.S., D.V.H.,

Officer in Charge of the Veterinary Laboratory, Wellington, New Zealand.

This disease is characterized by an acute inflammation of the skin covering the face, which may, if the affection be prolonged, become chronically thickened by the deposition of fibrous tissue. The affected parts become finally denuded of hair, and covered by a thick brown scab underrun with pus. The preliminary irritation, by which attention to the prevalence of the disease is first aroused, leads affected animals to attempt relief by vigorously rubbing the face and head against any available objects or on the ground-surface, or by violent scratching with the hind feet, and endeavouring by other forcible means to allay the irritation. The skin in consequence becomes abraded, and the inflammatory condition aggravated, while soil and dirt are introduced into the self-inflicted wounds. The inflammation extends, involving the tissues around the eyes, which receive further injury from the animal in the manner referred to: the resulting conjunctivitis and corneitis may produce blindness. It has been noticed that sheep affected with facial dermatitis are also frequently infested to a severe degree by stomach and intestinal worms. The irritation provoked by the presence of these parasites gives rise to

diarrhœa. This not infrequent combination of gastro-enteritis and dermatitis is often followed by death from malnutrition and exhaustion. The primary cause of this morbid condition appears to be due to some functional disorder of the digestive organs, followed, probably, by the absorption of toxins into the circulatory system. These either interfere with the cellular nutrition of the skin, or act as irritants, setting up an eczematous eruption.

Post-mortem examination of affected sheep reveals often a fatty and congested condition of the liver and kidneys, indicating that disturbance of the secretory functions of these organs has been taking place.

It has been remarked that sheep obliged to partake exclusively of one variety of food are more prone to be affected. Removal to fresh pastures, involving change of feed, generally checks the appearance of further cases, while, provided the disease has not advanced too far, and is not complicated with other disorders, affected animals recover. Recently, by the courtesy of owners, sheep suffering from facial dermatitis in various stages have been forwarded to the Laboratory for further study and observation regarding its nature. In every case examined, an organism corresponding to the *Staphylococcus citreus* has been recovered from the pustular eruption on the face. It has also been noticed that some necrosis of the skin and subcutaneous tissue accompanies the inflammatory lesion. In the depth of this necrosed tissue the presence of an organism identical with that of the necrosis bacillus has been observed.

Sheep inoculated experimentally with a pure culture of the staphylococcus have developed typical pustular eruptions, and the *Bacillus necrophorus* has been found in the lesion, associated with the coccus employed to inoculate the animal. I have also been able to produce identical lesions in the thigh of an experimental sheep by inoculation, after scarification, of an emulsion of its own dung. The ubiquitous nature of the necrosis organism has been referred to under the account of Bacterial Necrosis. There seems little reason to doubt that the same organism is implicated in promoting the pathological changes in facial dermatitis. It appears probable that the eczematous condition permits the cocci normally present on the skin to exercise their pyogenic properties, the necrosis bacillus gaining access to the diseased

tissues through the self-inflicted abrasions. In certain districts the larvæ of the sheep maggot-fly (*Œstrus ovis*) have been blamed for setting up this condition. Although not the direct cause, it may be assumed that the presence of the larvæ in the nasal cavities of sheep may be held in some cases partially responsible, the infected animal, by its efforts to allay the irritation produced by the parasite, injuring the facial tissues, and rendering them permeable to the *B. necrophorus*.

Cases Occurring Among Cattle.

Recently, Mr. Lyons, Veterinary Supervisor, Auckland District, has reported a similar condition affecting cattle. In these animals the skin-lesions are apparently not confined to the face, but may also involve the udder and perineum.

Considering how readily the parts affected may be soiled by dirt and droppings, the abraded cutaneous surface would provide a means of access to the necrosis organism, which may take a considerable share in the production of the lesions. I have not had an opportunity to personally observe the diseases attacking bovines.

Treatment.—The treatment of facial dermatitis has already been described in the Departmental Report, 1908, to which I have nothing further to add. Change of pasture on to a different variety of feed, and the application of an anodyne and antiseptic lotion—such as creolin or lysol—in fairly strong solution, usually promotes recovery. In the treatment of the disease in cattle, Mr. Lyons has found good effects result from local application of zinc ointment.

THE NEW OPERATION FOR ROARING—A QUESTION IN HISTORICAL ACCURACY.

By W. L. WILLIAMS,

Professor of Surgery in the New York State Veterinary College, Cornell University, Ithaca, N. Y.

There has recently appeared in the veterinary journals of England a series of articles in reference to a new, or old, operation for roaring, in connection with which the writer's name has been used, sometimes with credit, at others with discredit.

Hobday [8] says: "The extraordinary success which has,

however, followed a brief series of cases which have been operated upon by the method originally suggested by Dr. W. L. Williams. . . ."

Sutton [18] says: "The operation I wish to bring to your notice to-day is briefly described in Fleming's book as having been performed by the Günthers early in the nineteenth century. Why it fell into disuse it is hard to say. . . ."

Wooldridge [26] says: "This (*i.e.*, Günther's) is the operation we propose to perform to-day. It has lately been *re-introduced* by Professor W. L. Williams."

Woodruff [27] states: "It is not a *new* operation at all, for it was first performed and described by K. Günther, of Hanover, as a result of experiments which he began in 1845. Fleming says of this operation: 'Finally they (the Günthers, father and son) endeavoured to bring about a firm adhesion between the inner surface of the thyroid and the outer surface of the arytenoid cartilage by excising the laryngeal sac, but leaving the vocal cord intact.'

"Being uncertain in its results, the operation fell into disuse, but has recently been *revived*, and performed with considerable success by Professor W. L. Williams, of New York."

The editor of the VETERINARY NEWS [20] states: "Much interest has been aroused recently by a *revival* of some laryngeal operations for the relief of roaring"; and again, under the *nom de plume* of Verb. Sap. [21] says: "This was not a *new* operation, but simply a *revival* of an operation devised by the Günthers more than fifty years ago. . . ."; and again [22] says editorially: "The question has often been raised as to which term should be applied to a horse operated upon by Günther's method for roaring, and made able to gallop without 'making a noise.'"

Spicer [17] says: "The method employed was that initiated by Professor Woodruff"; and Robertson [16] says: "The late Principal Williams, in one or two instances, merely excised the mucous membrane of the left laryngeal ventricle, and I believe this operation was extensively performed by Mr. Edward Cotterill, surgeon, of Bicester."

The above statement by Hobday is faithful in every detail to a verbal communication made by the writer at the time when the operation was demonstrated to Hobday, in September, 1909.

The writer consequently assumes full responsibility for the statements of Hobday as to the origin of the operation, and will cheerfully answer to the veterinary profession for anything in that statement which is incorrect or unethical. So far as it appears in the periodical veterinary literature of England, the pure Americanism of this operation went unchallenged so long as it was considered a harmless farce, but as soon as it seriously threatened to be successful it changed its nationality like a fugitive from justice. Sutton, Wooldridge and Woodruff declare that it is of German origin; Spicer claims that it was "initiated" by an Englishman; and Robertson is sure that the credit belongs to a Scotchman.

Sutton [18], Wooldridge [26], and Woodruff [27] apparently depend upon the statement of Fleming [4], as quoted by Woodruff [27], as a basis for their assertions. Fleming [4] does not state, however, that Günther "introduced," "established," or "recommended" any such operation, but clearly otherwise; then he further says: "On the whole, the results obtained by the Professors Günther were unfavourable." While Fleming [4] says that Günther [5] excised the laryngeal sac and retained the vocal cord, he does not give the technique and offers no data which can in any way identify the experiment of Günther [5] with the operation now being performed in England.

The present status of the operation in England is conclusive proof of the non-identity with the experiment of Günther. When Hobday first made his announcement of the successful outcome in a few horses, a storm of criticism burst forth, seriously questioning the efficacy of the operation and predicting that ultimately it would share the fate of all preceding operations for roaring. Those doubts and criticisms are now stilled, and English veterinarians, as seen in the columns of the veterinary journals, admit that the operation is a success, and after only a year an astonishing number of horses are reported cured from roaring. A distinguished teacher of surgery in Great Britain assured the writer in 1909 that he had no faith in any operation for roaring; in 1910 a London instrument firm advertises the set of instruments for roaring as used by the eminent Professor. The degree of recovery is of a far higher order than has heretofore been recorded, so that now the hunter follows the hounds over hedge and ditch, and fen and moor, without betraying any

abnormal sound in breathing, which is certainly in marked contrast to practically all of the so-called cures which have heretofore been recorded. The operation has become so successful that, for the first time in the history of veterinary surgery, veterinarians and the editors of veterinary journals [21] are asking the question "If a horse is made sound by the new-old operation, is he sound or unsound?" Indeed the operation has become what might be termed *alarmingly successful*, and is taxing the ingenuity of some veterinarians to find a way foi detecting roaring where it does not exist. As a contrast, Günther [7] commenting upon the inadequate claim of Möller [13] that he had cured a majority of his cases by arytenectomy, though they still made a noise when exerted, says: "Das heisst doch mit anderen Worten; 'Vom Kehlkopfpfeifen sind sie gebeilt, nur darf man sic nicht untersuchen, sonst rohren sie!" (They are cured of roaring, only one dare not test them, lest they roar!) The energetic veterinary profession of Great Britain would not have permitted an operation of so great value to become dormant for half a century after it had once been introduced. Something new is occurring in England.

Cadiot [1]; Cadiot, translated by Dollar [2]; Dollar [3]; Hoffman [12;] Moeller [13]; Moeller, as translated into the French by Cadiot [14]; Vennerholm [19]; and all other writers we have been able to consult, agree with Fleming that Günther's work was confined to experimental operations, which practically always failed and were never applied clinically, and that Günther did not *establish*, *introduce*, or *recommend* any form of operation for roaring.

A footnote on page 137 of Fleming's work [4] indicates that his statement is based on a footnote on page 96 of Günther's *Myologie* [5], which reads: "Die Stimmtasche zwischen Schild- und Giesskannenknorpel entfernt, aber das Stimmband geschont (der hinter Rand des Giesskannenknorpels wurde dabei regelmässig bis nahe unter die Artikulationsfläche des Giesskannenknorpels von der Kehikopfwand getrennt, so dass er aufwarts nur durch die Kehlkopfsschleimhaut und abwarts durch das verdünnte Stimmband mit dem Schildknorpel in Verbindung blieb; in einzelnen Fällen heilte die aussere Flache des Giesshannenknorpels sehr gut an den Schildkronpel fest, und die Pferde waren und blieben geheilt, in andern Fällen heilte der

Knorpel zu niedrig an und die Thiere blieben Rohrer, in noch andern Fällen heilte der Giesskannenknorpel nicht fest genug an und die Thiere rohrten mit schlodderndem Geräusch." Günther offers no suggestion that he established, introduced, advised, or recommended the operation under discussion. He does not give in detail his technique, but says enough to positively and finally establish the total non-identity of his experimental operation with the operation which the writer demonstrated to Mr. Hobday. When Günther states that the posterior border of the arytenoid cartilage was separated, in the operation, from the thyroid until immediately beneath the articular surface of the arytenoid, so that the arytenoid cartilage remained in connection with the thyroid cartilage above, only through the medium of the laryngeal mucosa and downward by the thinned vocal cord, it becomes very clear that the technique of Günther bore no resemblance whatever to the operation now in use. The latter part of the quoted paragraph shows that the technique was radically different, since otherwise the results recorded by Günther could not have occurred. Conclusive evidence that their work was confined to experiment animals is found in the following paragraph from the same footnote [5]: "Die Versuche sind sehr kostspielig, da nur gute, kräftige Pferde für den Zweck zu verwenden sind, und diese zugleich längere Zeit hindurch gut gefüttert werden mussen. Ich habe von Seiten unserer Anstalt keinerlei Subventionen zu dènselben erhalten, habe sie vielmehr mit einem Kostenaufwande von p.m. 600 Thlrn. ausgefürt."

In the first edition of Günther's monograph upon roaring [6] he states on page 50: "Wegen der Unsicherheit des Erfolges habe ich meine Operationsmethoden niemals empfohlen und habe sie deshalb auch nich von den Studirenden einuben lassen." In his second edition [7], 1896, he repeats this statement, and upon the last page says: "Es ergibt sich sonach, das ein operatives Verfahren auf den bisherigen Wegen nicht zum Ziele führt." He then states that tracheotomy affords the only satisfactory relief.

Thus K. Günther declares over and over again that he never introduced, established, or recommended any operation for roaring, and does not intimate that he ever operated clinically upon a roarer.

The writer has no desire to detract from the fame of the

Professors Günther, who worked for more than half a century upon this problem with unflagging zeal without reaching their goal. They insisted that, if roaring were to be surgically relieved, it must be through some operation upon the arytenoid cartilages, vocal cords, or laryngeal ventricles, and they experimented variously with each. Cadiot, Fleming, Hoffman, Moeller, Vennerholm, and all subsequent investigators worked over the same field which the Günthers mapped out. If finally the riddle has been solved, the solution is a tribute to their labours, a far greater tribute than a declaration that they introduced an operation, which the Günthers themselves emphatically deny.

One other question is raised in the articles mentioned, especially when "Perplexed" [15] says: "At each re-appearance (of operations for roaring), history repeats itself—first a little secrecy, next a boom and a rush, and then a gentle fizzle out." "Perplexed," under the secrecy of a *nom de plume*, hints at unprofessional secrecy in regard to the introduction of the operation into England. So far as the writer knows, Mr. Hobday did not advertise the demonstrations; neither were they done in secret, but in broad daylight, close beside a public highway, with the gates of the enclosure standing open. So far as the writer knew them, or English veterinary journals have indicated since, Mr. Hobday had invited all the veterinarians in England who cared, at that time, to see the operation. It would have been offensive to many English veterinarians to have invited them to witness Günther's operation for roaring, by an unknown American who had never met Günther or communicated with him, and who, at that time, had never seen any one of Günther's works, and consequently knew nothing whatever of Günther's operations.

In a yet broader sense, the operation was not secret. The writer reported his investigation, with clinical statistics, in 1906, to the American Veterinary Medical Association [23]; an amended technique in 1907 [24]; and again, in 1907 [25], the technic was published in the writer's handbook, which was offered to English veterinarians by a leading London publisher, upon whose shelves the book has attracted more dust than attention.

The operation and its results had been the common property of the veterinary profession for three years when the demonstra

tion was made in London, and if it still remained a secret to "Perplexed" the cause lay at his own door. During the summer of 1909, the writer freely explained the technique and results of the operation to numerous English veterinarians. One man in England (Hobday) had confidence in the operator and operation, and his request for a demonstration was granted with the same courtesy that has always awaited such requests of the writer from any reputable veterinarian, at home or abroad.

The current literature of to-day becomes history to-morrow, and, in behalf of historical accuracy, it is hoped that the English veterinary journals will carefully investigate the source of origin of the operation for roaring now being performed in England, and place the facts clearly on record.

LITERATURE CONSULTED.

[1] CADIOT, P. J. "Traitement Chirurgical du Cornage Chronique," 1891.

[2] *Idem.* "Roaring in Horses," translated by T. J. W. Dollar, 1892.

[3] DOLLAR, J. A. W. "Roaring in Horses." A translation of P. J. Cadiot's "Traitement Chirurgical du Cornage Chronique," 1892.

[4] FLEMING, GEORGE. "Roaring in Horses." 1889.

[5] GÜNTHER, K. "Topographische Myologie des Pferdes," 1866.

[6] *Idem.* "Studien uber Kehlkopfpfeifen der Pferde," 1893.

[7] *Idem.* "Studien uber das Kehlkopfpfeifen der Pferde." Zweite Auflage, 1896.

[8] HOBDAY, FREDERICK. "A Preliminary Note upon the New Operation for Roaring in Horses." VETERINARY JOURNAL, January, 1910. Vol. 66, p. 23.

[9] *Idem.* "The Newly-introduced Roaring Operation." *Veterinary Record*, April 16, 1910. Vol. xxii., p. 692.

[10] *Idem.* "The Newly-introduced Roaring Operation: an Improvement and a Warning." VETERINARY JOURNAL, August, 1910. Vol. 66, p. 487.

[11] *Idem.* "Some Further Remarks on the Roaring Operation." *Veterinary Record*, October 29, 1910. Vol. xxiii., p. 276.

[12] HOFFMAN, Professor L. "Tierarztliche Chirurgie." Band i., 1892.

[13] MOELLER, H. "Das Kehlkopfpfeifen der Pferde und seine Operative Behandlung." 1888.

[14] *Idem.* Translated into French by P. J. Cadiot. "Le Cornage laryngien chez le cheval (hemiplegie laryngienne) et son traitement chirurgical." *Annales de Méd. Vét.*, 1888-1889. Vol. xxxviii., p. 79.

[15] "Perplexed." "Operations for Roaring." *Veterinary Record*, November 26, 1910. Vol. xxiii., p. 338.

[16] ROBERTSON, J. B. "The Operative Treatment for Roaring." *Veterinary Record*, July 2, 1910. Vol. xxiii., p. 3.

[17] SPICER, ARNOLD. "New Operation for Roaring." *Veterinary Record*, June 18, 1910. Vol. xxii., p. 836.

[18] SUTTON, GUY. "Roaring in Horses." *Veterinary Record*, May 7, 1910. Vol. xxii., p. 729.
[19] VENNERHOLM, Professor Dr. JOHN. "Spezielle Operations lehre des Pferdes," 1907, p. 175.
[20] *Veterinary News.* "Operations for Roaring in Horses." Editorial. March 26, 1910. Vol. vii., p. 171.
[21] *Idem.* Editorial, *Verb. Sap.* "The Operation for the Relief of Roaring." April 30, 1910. Vol. vii., p. 236.
[22] *Idem.* Editorial. "Sound or Unsound." November 5, 1910. Vol. vii., p. 627.
[23] WILLIAMS, W. L. "Notes on the Surgical Relief of Roaring." *Proceedings of the American Veterinary Medical Association*, 1906, p. 179.
[24] *Idem.* "The Surgical Relief of Roaring." *Proceedings of the American Veterinary Medical Association*, 1907, p. 430.
[25] *Idem.* "Surgical and Obstetrical Operations," 1907.
[26] WOOLDRIDGE, GEO. H. "The Relief of Roaring in Horses by Excision of the Left Laryngeal Ventricle." VETERINARY JOURNAL, May, 1910. Vol. lxvi., p. 250; *Veterinary Record*, June 11, 1910, Vol. xxii., p. 811; *Veterinary News*, June 18, 1910. Vol. vii., p. 335.
[27] WOODRUFF, HAROLD A. "The Operative Treatment of Roaring in Horses." *Veterinary Record*, March 19, 1910, Vol. xxii., p. 620; *Veterinary News*, March 26, 1910, Vol. vii., p. 164.

EXPERIMENTS TO ASCERTAIN IF CATTLE MAY ACT AS A RESERVOIR OF THE VIRUS OF SLEEPING SICKNESS (*TRYPANOSOMA GAMBIENSE*).*

BY COLONEL SIR DAVID BRUCE, C.B., F.R.S.,
CAPTAINS A. E. HAMERTON, D.S.O., AND H. R. BATEMAN,
Royal Army Medical Corps;
AND
CAPTAIN F. P. MACKIE,
Indian Medical Service.

Indexed B. A. L.

THE question as to whether cattle can act as a reservoir of the virus of sleeping sickness is an important one. It was usually believed until lately that man was the main reservoir, and that the other animals might be ignored. But in view of the fact that the flies on the Lake-shore have remained infective for some two years after the native population have been removed, it is necessary to inquire if it is not possible that other animals may act as well.

In this regard cattle have been, perhaps, the most important, as on the once thickly populated Lake-shore and islands they were numerous, and in many cases grazed and watered in the fly-area. Another reason of their importance is, that if they can act as a reservoir, then the

* Reprinted from the *Proceedings of the Royal Society*, B., vol. lxxxii. 1910.

same will probably be true of the different species of antelope which inhabit the Lake-shore. It may be presumed that these will greatly increase now that the natives and domestic animals have been removed, and that they will take the place of the cattle in keeping up the infectivity of the *Glossina palpalis*.

The Commission, therefore, thought it would be well to inquire into the question, and the result is given in this paper.

Answers to the following questions were sought: Are cattle capable of being infected with sleeping sickness by the subcutaneous injection of blood containing *Trypanosoma gambiense?* Can cattle be infected with sleeping sickness by the bites of artificially infected tsetse-flies? Can cattle be infected with sleeping sickness by the bites of the naturally infected flies caught on the Lake-shore? Is it possible to infect tsetse-flies by feeding them on cattle infected with sleeping sickness, and afterwards to transmit the disease by means of these flies to healthy animals? Finally, if these questions are answered in the affirmative, will it be possible to find that cattle which have lived in the fly-area are naturally infected with sleeping sickness?

(I.) Are Cattle capable of being Infected with Sleeping Sickness by Subcutaneous Injection of Blood containing *Trypanosoma gambiense?*

Experiment 869. *Bull.*

September 10, 1909.—A bull was inoculated with 5 cc. of blood containing large numbers of *T. gambiense* from an infected monkey.

Its blood was examined daily, and 18 days after injection the bull was found to be infected with *T. gambiense*. The identity of the trypanosome was established by injecting a monkey with some blood from the ox. This monkey showed *T. gambiense* on the sixth day.

Conclusion.—From this experiment it is seen that oxen are capable of being infected with sleeping sickness by the injection of blood containing *T. gambiense*. The trypanosome appears in small numbers in the blood, and the blood, when injected into susceptible animals such as monkeys, gives rise to a fatal form of the disease.

(II.) Can Cattle be Infected with Sleeping Sickness by the Bites of Artificially Infected *Glossina palpalis?*

The two following experiments were carried out by feeding *G. palpalis* first on an infected monkey, and immediately afterwards on a healthy ox. Wild flies from the Lake-shore were used.

Experiment 890. *Ox.*

May 20, 1909.—The ox was thrown and a monkey heavily infected with sleeping sickness was laid across its flank. Two cages of *G. palpalis*, containing 100 and 150 flies respectively, were allowed to feed for a few seconds on the monkey and then on the ox. The flies were allowed from 30 to 35 interrupted feeds on each animal every day. This was continued for 38 days, during which time 561 flies were estimated to have fed on one or other animal.

July 17.—Fifty-eight days after the first infected feed *Trypanosoma gambiense* appeared in the blood of the ox.

The identity of the trypanosome was established by injection of the ox's blood into two monkeys. The first monkey was injected with blood from the ox 76 days, and the second monkey 181 days after the flies had first fed on the ox. Both monkeys developed sleeping sickness, the first seven days and the second 11 days after injection of the blood.

Experiment 891. *Calf.*

The details of this experiment were similar to those of the last. *T. gambiense* appeared in the blood of the calf 57 days after the flies had been first fed upon it.

Three cubic centimetres of the blood of the calf were injected into a monkey, and the monkey developed sleeping sickness after an incubation period of eight days.

Conclusion.—These two experiments show that when artificially infected *G. palpalis* are allowed to feed on healthy cattle, these animals develop sleeping sickness, and that the blood of the cattle is capable of giving rise to infection of *T. gambiense* in monkeys when injected into them.

(III.)—Can Cattle be Infected with Sleeping Sickness by the Bites of the Naturally Infected Flies caught on the Lake-shore?

In the next three experiments freshly caught *G. palpalis* brought up to the laboratory from the Lake-shore were allowed to feed straightway on healthy cattle. By this means it will be shown whether *G. palpalis* in their wild state are capable of giving sleeping sickness to healthy cattle.

Experiment 982. *Bull.*

2,195 freshly captured *G palpalis* were applied to a bull, and of these 1,536 were estimated to have fed. The feeding of the flies

extended over a period of 16 days, at the end of which time *T. gambiense* appeared in the blood of the bull.

To help in the identification of this trypanosome 3 cc. of the blood of the bull were injected into a monkey. The monkey developed sleeping sickness 18 days later. 5 cc. of the blood of the bull were also injected into a goat. *T. gambiense* appeared in the blood of the goat after an incubation period of 38 days.

Experiment 1,462. *Bull.*

The details of this experiment were similar to those of the last one. Over a period of eight days 1,370 wild flies from the Lake-shore were applied to the bull, of which 705 fed. Ten days from the first application of flies *T. gambiense* appeared in the blood of the bull.

Two animals, a monkey and a goat, each received 1 cc. of the blood of the bull by injection under their skin. The monkey developed sleeping sickness seven days later, but the goat died in 16 days without showing any infection.

Experiment 1,465. *Bull.*

During a period of 13 days, 459 freshly caught Lake-shore *G. palpalis* were applied to a bull, and of these 314 fed. On the 14th day after the flies were first fed the bull developed an infection of *T. gambiense.*

Some blood from this bull was injected into a monkey and into a goat. Neither animal became infected.

Conclusion.—These experiments prove that *G. palpalis*, when captured in their natural state on the Lake-shore, are capable of transmitting the virus of sleeping sickness to cattle, and that the blood of these cattle gives rise to a fatal form of the disease in monkeys and goats when it is injected into them.

(IV.)—Is it possible to Infect Tsetse-flies by Feeding them on Cattle Infected with Sleeping Sickness, and afterwards to Transmit the Disease by means of these Flies to Healthy Animals?

Five experiments nnder this heading were carried out. Laboratory-bred flies were used in all of them. Three were negative and two positive. The three negative experiments will be shortly summarized first.

Experiment 1,451.

Ninety laboratory-bred *G. palpalis* were fed for ten successive days on a calf whose blood contained *T. gambiense.* The flies were starved

for 72 hours. They were then fed on a clean monkey daily for 45 successive days. The monkey failed to develop sleeping sickness.

When the remainder of the flies were dissected, one contained flagellates, but when the contents of this fly were injected into a goat the animal failed to show any infection of *T. gambiense.*

Result.—Negative.

Experiment 1,269.

The details of this experiment were similar to those of the last After the *G. palpalis* had been fed on two oxen whose blood contained *T. gambiense,* they were applied daily to a monkey. They were fed on this monkey for 35 consecutive days and were then transferred to a second monkey. Both the monkeys remained healthy.

Two of the flies were found on dissection to contain flagellates, but when these were injected into a monkey and a goat no development of sleeping sickness took place in these animals.

Result.—Negative.

Experiment 1,672.

Here again the technique was similar to the last. The *G. palpalis* were fed on alternate days for a lengthened period, on a clean monkey and a clean goat. Both animals remained healthy.

Some infected flies were found on dissection, but when introduced under the skin of a goat and of a monkey did not give rise to sleeping sickness.

Result.—Negative.

The next two experiments, which were carried out in the same way as the two preceding ones, were positive.

Experiment 1,566.

The *G. palpalis* were fed on an infected ox, and after a starve of 72 hours were fed on a clean monkey for 45 successive days. Sixty-eight days after the flies had taken their first infected feed this monkey developed sleeping sickness.

When the flies came to be dissected nine of them showed flagellates either in the proboscis or in the alimentary tract. Some of these were injected into goats and into a monkey, but with negative results.

Result.—Positive.

Experiment 1,602.

Fifty laboratory-bred flies were fed for four successive days on an ox whose blood contained *T. gambiense.* After a period of starvation

they were applied to a monkey and to a goat on alternate days. The monkey died before it could have become infected, but the goat developed sleeping sickness 20 days after the flies had their first infected feed.

The remainder of the flies, 32 in all, were dissected, and five were found to contain flagellates. The alimentary contents of one of these flies were injected into a monkey, and after an incubation period of 13 days *T. gambiense* appeared in its blood.

Result.—Positive.

Conclusion.—Laboratory-bred tsetse-flies can be infected by feeding them on cattle infected with sleeping sickness, and afterwards the disease can be transmitted to healthy animals by means of these flies.

(V.) Do Cattle, when Living in the Fly-area, actually carry the Virus of Sleeping Sickness?

About seventeen cattle from various sources were examined with this point in view. Not all these cattle could be proved to have been exposed to the bites of *G. palpalis*, but most of them came from places where these flies are plentiful. One was positive.

Experiment 1,633.

This cow came from the island of Kome, in Lake Victoria, where human sleeping sickness is prevalent and where *G. palpalis* abound.

T. gambiense was found in its blood by microscopical examination, and when 3 cc. of the blood were injected under the skin of a monkey the animal developed sleeping sickness after an incubation period of seven days.

Conclusion.—This experiment proves that cattle in their natural state, and apparently in good health, may harbour the virus of sleeping sickness.

General Conclusions.

It has been proved by experiment that cattle may act as a reservoir of the virus of sleeping sickness, and that healthy animals may be infected from them by means of *G. palpalis.*

It has also been proved that cattle in the fly-area do naturally harbour *G. gambiense.*

It is, therefore, possible that the cattle and antelope living in the fly-area may act as a reservoir, and so keep up the infectivity of the *G. palpalis* for an indefinite period, but there is no proof up to the present that this actually takes place in Nature.—*Journal of the Royal Army Medical Corps.*

RABIES AND ITS CONTROL IN INDIA.

By Captain E. CLIVE WEBB, F.R.C.V.S.,

Indexed B. A. I. *Army Veterinary Corps, Aldershot.*

Rabies is an acute specific disease to which all mammals are susceptible, although it affects more particularly the canine species. In most countries its continued existence is bound up with this species, and although the disease is common amongst certain wild animals (in India—jackals), it is probable that the dog is the natural reservoir, and if the disease were stamped out in the latter species it would probably also die out in the wild animals in which it is known to exist at present.

Etiology.—The causal organism of the disease has not yet been discovered and hence it is customary to speak about the "virus" of rabies, although it is now universally admitted that the cause of the disease is an organism, and this latter must be an obligatory parasite since the disease never occurs spontaneously. Rabies therefore belongs to the group of diseases caused by the "ultra-visible viruses."

Remlinger,* in 1903, showed that the virus of rabies was filtrable, although prior to this year such was not believed to be the case. He found that the virus of rabies failed to traverse the close-grained Chamberland B and F filters, but proved that under certain conditions the virus is not arrested by the more open-textured Berkefeld filter.

Remlinger partly confirmed the results of earlier observers, and his results also were subsequently confirmed by still other observers (Betarelli-Maggei). Knowledge of the above facts might be usefully employed in endeavouring to arrive at a diagnosis from brain material, which has become too putrid to inoculate without previous filtration. The open-textured Berkefeld filter would arrest putrefactive organisms, whilst allowing the rabies virus to pass. By using such a filtrate for inoculation experiments, the danger of the experimental animals dying from septicæmia before the result of the inoculation of the rabies virus, if present, could be observed, would be obviated. Up to the present time it has not been found possible to cultivate the rabies virus artificially.

Method of Infection.—The natural method of infection is by

* *Annales de l'Institute Pasteur*, 1903.

inoculation, the inoculating agent being almost invariably the tooth of a rabid dog, the inoculated material being saliva. This is the only natural method of infection. It is possible, however, for infection to be brought about by the application of saliva to an abraded surface. Hence the danger of allowing oneself to be licked by a dog, even should the dog, to all outward appearances, seem healthy. However, I shall refer again to this point later.

Distribution of the Virus in the Body.—The virus is found in the central nervous system, the medulla oblongata being its chief seat of propagation. It might here be remarked that when forwarding a piece of brain from an animal suspected of rabies for examination and report, it is best to select the piece from the back part of the brain.

The virus is also abundantly present in the salivary secretion.

The peripheral nerves also sometimes contain the virus, and it is also sometimes present in other secretions than the salivary —*e.g.*, the milk of rabid animals is sometimes infective. The blood is non-infective.

Rabies differs from the majority of other microbic diseases in the way in which the causal virus travels to the central nervous system when inoculated in any dependent part of the body —viz., by way of the nerve trunks and not by way of the vascular or lymphatic circulation.

This is an important factor to be remembered with regard to rabies for the following reasons: The further away from the central nervous system that a bite is inflicted the longer is the period of incubation, and this point is not lost sight of in applying antirabic treatment. Patients who have been bitten about the face or neck are submitted to a more drastic treatment than those bitten in parts further removed from the central nervous system. The treatment is in these former cases made more drastic by the use of vaccine material, the virus of which has been less attenuated than is the case with the ordinary vaccine used.

Period of Incubation varies within wide limits. May be taken as roughly between three weeks and six months, although cases are on record of the period of incubation running into a year or more, and it has been said that there have been cases in the human subject in which the period of incubation has been several years.

However, the fact of a dog having been bitten by a rabid dog a year prior to its developing clinical symptoms of rabies is no proof whatever of that period being the period of incubation, since it is impossible to keep a dog under such close observation for so long a period, so that one could affirm that it had not been bitten again at a later date. From personal observation I should be more inclined to favour this latter supposition, especially if such a case occurred in a country rife with the disease, and should view with grave doubt any report of such a long period of incubation having occurred unless the proof of it were incontrovertible. For practical purposes I consider that six months is a safe period to allow as the longest probable period of incubation.

Symptoms.—Rabies in dogs is manifested in two different forms, which are distinguished as furious and dumb rabies.

Furious Rabies.—The premonitory symptoms of this form, as well as of the dumb form, are more or less identical. The first noticeable symptom is a change in the disposition of the animal. This first change is not characterized by a disposition to bite; an the contrary, it is usually manifested by dulness and apathy. The lively dog becomes dull and listless, and the usually unexcitable, undemonstrative animal may become more affectionate and show a disposition to fawn upon its owner. The noisy dog becomes perhaps suddenly quiet, whereas a usually quiet dog may take to growling without apparent cause. These slight variations in the normal disposition of a healthy dog can be enlarged upon *ad infinitum*, but to sum them up—any sudden and capricious change in the disposition of a dog in a country where rabies is common should at once be viewed with strong suspicion, and steps should immediately be taken to segregate the animal until a definite diagnosis can be made. Nervousness, manifested by increased sensibility to sound and restlessness, is very suspicious of rabies. A depraved appetite is an almost invariable symptom. A rabid dog will swallow all sorts of things foreign to his usual tastes—*e.g.*, straw, bits of string, paper, stones, &c., &c., and the presence of such bodies in the stomach in *post-mortem* examination of a suspected case is very strong evidence in support of the diagnosis rabies. It is said that rabid dogs have a disposition to bay the moon and that their voice has a peculiar sound. Personally I have never observed either of these phenomena, and should pay little attention to such as

evidence in support of the disease being rabies. In these early stages the dog still recognizes his master's voice and will obey it, although sullenly and without the usual pleasure and promptness. The presence of wounds which may have been caused by bites can hardly be considered of importance, since few healthy dogs are ever whole-skinned; although, on the other hand, were there a history of the patient having been attacked by a strange dog with resulting wounds, such would then help to swell the evidence. Mental disturbance is usually manifested at this early stage by a vacant expression and snapping at imaginary objects, although the animal may temporarily rally at the sound of a human voice. These nervous symptoms (hallucinations) are intensified as the disease progresses, the patient becomes more restless, reflex excitability and hyperæsthesia are increased, and the animal now becomes aggressive, will bite at any object held up to him, and finally, if at large, will dash off and gallop for miles, attacking any animal or human being who may cross his path. He may finally return to his old haunts exhausted or succumb from exhaustion during the course of his mad career. It is said that a rabid dog will not go out of his way to attack a man or animal, but will only do so should they be in his way. It is safer to assume that a rabid dog may take it into its head to make an unprovoked attack on any living being that may be within the range of its vision, although it is true that the dog's tendency is to get forward without waste of time. I have personally seen a hound bitch deviate from her course to attack a buffalo and other dogs. The rabid dog, however, does not appear to wish to fight it out to a finish, but having bowled over and bitten at its imagined enemy, it continues on its way. Its attack also is silent. These furious symptoms are followed by debility, paralysis, and death. An occasional and characteristic symptom of the furious form of rabies, both in dogs and other animals, is their biting of themselves, and they will frequently lacerate themselves deeply about the breast and forearms.

Dumb Rabies.—In this form of rabies there are no preliminary furious paroxysms. The premonitory symptoms of dulness and altered disposition are the same as in the furious form, but these symptoms are rapidly succeeded by paralysis. Usually the first muscles to become paralyzed are the masseters, as a result of which the lower jaw drops and there is a dribbling of saliva.

There is neither the inclination nor ability to bite. The patient quickly becomes debilitated and emaciated owing to inability to swallow. Paralysis of the hind-quarters usually follow, then prostration and death. Paralysis may commence elsewhere than in the face, but the dropped jaw and drivelling saliva is almost pathognomonic. I have seen many cases of this kind in imported foxhounds in India, and, in fact, in the hills during the hot weather months it appears to be quite as common, if not more so, than the furious form. One may meet with other cases intermediary, so to speak, between the dumb and furious forms.

Diagnosis.—The recognition of rabies in its earliest stages is of the utmost importance, and owing to the danger of spreading the disease which may result from allowing a case to go unrecognized and not taking the necessary precautions, one cannot err on the side of caution.

Should the disposition of a dog become suddenly altered and there is added to this a history of its having been bitten, suspicions should at once be aroused. The later symptoms detailed above enable one to form almost with certainty a correct diagnosis. Any dog, whose habits are usually docile, that makes an unprovoked attack on any human being should at once be suspected.

Having detailed the usual symptoms manifested, it must be added that there are no means of making a positive diagnosis *ante-mortem*, but in the majority of cases the manifestation of any or all of these symptoms, together with certain *post-mortem* appearances to be detailed later, enable one to arrive at a correct diagnosis.

Post-mortem Appearances—Macroscopic.—Congestion of the mucous membrane; absence of normal food-material in the stomach; presence of foreign bodies in the stomach (pieces of straw, stick, rags, stones, &c.). Sometimes the stomach is completely empty, excepting for a little brown-tinged mucus.

The above are usually the only macroscopic lesions observable which are at all diagnostic.

To sum up—the absence of ordinary food material and the presence of considerable quantities of foreign materials in the stomach of a dog that has made an unprovoked attack on a human being leave hardly any room for doubt that the animal was actually rabid. On the other hand, the presence of a con-

siderable quantity of ordinary food is rather strong evidence against the existence of rabies.

Microscopic Lesions.—In 1886 Babes* concluded, as a result of numerous researches in man and dogs, that the essential lesion of rabies consisted in an accumulation of embryonic cells in the neighbourhood of the central canal, and especially about the large modified cells of the motor centres of the bulb and cord. In 1892 he reaffirmed his observations, and held that it was possible to make a rapid diagnosis of the disease by a microscopic examination of the bulb and cord. He described in the bulb what he considered the diagnostic lesion of the disease, viz., pericellular accumulations of embryonal cells. The cells of the bulbar nuclei undergo degeneration and present various stages of chromatolysis. There is a loss of the prolongations and a progressive modification and even total disappearance of the nuclei, a dilatation of the pericellular space, and an invasion not only of this space, but also of the nerve-cells and embryonal cells, and at the same time small corpuscles which are hyaline, brownish, and in parts metachromatic. Many of the nerve cells become surrounded by a large zone of embryonal cells, and when the cell is completely degenerated these occupy the cell area and constitute the rabic tubercle. Nélis and Van Gehuchten first noted what they considered to be a more diagnostic lesion of rabies than any other—*i.e.*, the most profound, constant, and earliest lesions are noted in the peripheral, cerebral, and sympathetic ganglia, and the changes are specially marked in the intervertebral ganglia and in the plexiform ganglia of the pneumogastric nerve. The characteristic changes consist in the atrophy invasion and the destruction of the nerve cells brought about by new-formed cells derived from the capsule which appear between the cell body and its endothelial capsule. These new-formed cells invade the nerve cell and finally occupy its entire capsule.

Cuillé and Vallée found that in animals in which the disease had just become manifest the lesions in the plexiform ganglia were slight or absent. They concluded that as a means of diagnosis the method of Van Gehuchten and Nélis has great value in the case of animals in which the disease has run its full course, ending in death.

* *Journ. of Comp. Path. and Tech.*, Vol. xiv., p. 37, "The Rapid Diagnosis of Rabies," by Mazyck P. Ravenel, M.D., and J. McCarthy, M.D.

The work of Nélis and Van Gehuchten was confirmed by Ravenel and McCarthy—purely contributory evidence. Cerebral ganglia show the most marked lesions, notably that of the pneumogastric nerve.

Rabieaux* also confirmed these operations. The experience of Rabieaux and of all the other authors who have investigated the subject shows that the practical value of this method of diagnosis varies according to the nature of the case. If the suspected animal has been killed at an early stage of the disease its value is very restricted, for the lesions are then at the most very slight and most frequently entirely absent. Consequently the non-detection of the lesions never justifies one in pronouncing the case not rabies. On the other hand, in the case of animals that have died, the before-described lesions are constantly present, and such lesions have not hitherto been observed with certainty in animals dead from other diseases than rabies.

In March, 1903, Négri announced that in many nerve cells obtained from rabid animals it was possible to detect corpuscles having special staining properties. These Négri described as the parasite of rabies. They appear as oval, rounded, or kidney-shaped bodies varying from 5 to 27 or 30 microns in diameter, lodged in the protoplasm close to the nucleus. These corpuscles are strongly refractile, and appear to contain granules in their interior. The number in different cells varies, and from ten to fifteen may be present in one cell. These corpuscles can be found in all parts of the nervous system, but are most numerous and constant in the large pyramidal cells of the hippocampus. They exhibit special staining properties. Négri held that the presence of these corpuscles afforded a rapid method of diagnosis. He admitted, however, that in rare cases these characteristic corpuscles could not be demonstrated, although the animal might be proved to be rabid. Other authors have confirmed Négri's statements, and all are agreed that the corpuscles can only be found in rabid animals.

Forgeot and Nicolas† investigated this subject and came to the following conclusions:—

(*a*) The corpuscles are never found except in cases of rabies.

(*b*) When found in the hippocampus of a suspected animal a positive diagnosis can be made.

* *Journal de Méd. Vét.*, December, 1902.

† *Journal de Méd. Vét. et de Zootech.*, September, 1905.

In an article entitled "The Etiology and Diagnosis of Hydrophobia" (*Journal of Infectious Diseases*, vol. iii., 1906), Anna W. Williams and May M. Lowden confirm the above observations, but state also as follows:—"The significance of the bodies is still in doubt for the following reasons: (*a*) They have not been found in all cases of hydrophobia, notably not in the fixed virus, nor in all parts of the nervous tissue proved to be virulent; (*b*) forms small enough to pass through the coarser Berkefeld filters have not been seen; (*c*) the structure has not been definitely shown to be analogous to that of known living organisms; (d) no definite series of forms indicating growth and multiplication have been demonstrated."

Evidence, therefore, is against these bodies being living parasites.

From the foregoing remarks on the nervous lesions it will be gathered that both the rabic tubercles of Babes and the corpuscles of Négri form valuable aids to diagnosis, but that negative results of histological examinations do not justify a verdict of not rabies.

The only absolutely certain method of proving whether or not an animal was rabid is by experimental inoculation. The material inoculated is a piece of spinal cord or medulla—preferably the latter—from the suspected animal, and the rabbit is the animal selected for the inoculation. A small quantity of an emulsion in bouillon or water of the brain of the suspected animal is inoculated subdurally after trephining into not less than three rabbits. Strict aseptic precautions are necessary. Provided that the inoculated rabbits survive a sufficient length of time—*i.e.*, the usual period of incubation of fourteen or fifteen days—and that the dog from which the brain material used for the inoculation was the subject of rabies, the inoculated rabbits will infallibly develop rabies (McFadyean).

The period of incubation in the rabbit inoculated with brain material from a natural case of rabies in the dog is usually about fourteen or fifteen days, and the period of visible illness one or two days.

The symptoms are those of gradual paralysis, commencing usually in the hind-quarters and rapidly extending forwards. As a rule, there is no cerebral disturbance and there is no propensity to bite.

Possible causes of failure are in cases in which the brain used for inoculation is partially putrid. In these cases the putrefactive organisms will almost certainly cause the death of the inoculated rabbit from a local inflammation of the membranes of the brain within a few days, whereas for the success of the operation the rabbits inoculated must survive at least a fortnight, and also must not be the subject of any other cererbal disturbance.

For the above reason, when forwarding a portion of brain material of a suspected case to a laboratory for inoculation purposes, it should be placed in pure glycerine. This latter substance possesses a double advantage. It can preserve the virus of rabies for about a month and a number of putrefactive organisms lose their vitality when immersed in it.

Treatment.—There is no treatment for dogs the subject of rabies, and even if there were any drug known to be efficacious in a proportion of cases, treatment would never be justified owing to the great danger of keeping a rabid animal alive, a danger both to the attendant and, should the patient by some accident get at large, to other animals and human beings.

A dog showing all the clinical symptoms of rabies should be immediately destroyed and the carcase burnt.

There are certain cases, however, in which it is very necessary to have the diagnosis confirmed, either microscopically or by experimental inoculation. The following are such cases:—

(*a*) Should the dog be known to have bitten other dogs, their confirmation is necessary in order that prophylactic measures may be taken with regard to the dogs bitten. Reference will be made to these measures later.

(*b*) Should any human being have been bitten or licked on an abrasion by a dog suspected of rabies, confirmation of the diagnosis should also be obtained. The reason of this is obvious, viz., the person bitten is relieved of anxiety should it be proved that the case was not one of rabies, and if the said person were already undergoing antirabic treatment the latter would be immediately stopped.

Therefore if there is no evidence forthcoming that any human being or other animal has been bitten by a dog destroyed on account of its having shown clinical symptoms of rabies, one is justified in allowing the diagnosis to rest on the latter, and no further precaution need be taken. If such evidence, however, is

forthcoming, then material should be forwarded at once to the nearest laboratory, with the history of the case, for the latter to be confirmed or otherwise. The procedure is as follows:—

It has already been pointed out that there are two methods of diagnosing rabies *post-mortem*, viz., microscopical and experimental. Brain material is necessary for both these methods of examination. When destroying the dog, therefore, the cranium should be mutilated as little as possible.

Major George Lamb, M.D., I.M.S., Director Pasteur Institute, Kasauli, in his pamphlet entitled "Rabies and Antirabic Treatment in India," August 1, 1908, gives the following instructions for the removal and getting up for despatch the brain of a supposed rabid animal:—

"The first procedure, therefore, is to open the skull and expose the brain. First wash the head well with an antiseptic such as carbolic acid, phenyle, &c. Then take a hammer and with a few hard sharp blows fracture into many pieces through the intact skin the top and sides of the brain cavity. With a knife throw back the skin and then remove as carefully as possible the pieces of fractured bone, in this way exposing the brain. For the experimental method with a clean knife remove a small piece of brain about the size of a bean and place it in a small bottle of pure glycerine. On no account is an antiseptic to be allowed to come in contact with the piece of brain, nor is anything to be added to the glycerine.

"For the microscopical method a special portion of the brain is desirable, viz., the hippocampus major. As this is somewhat difficult to dissect out by anyone unaccustomed to such work, it is best to remove the brain entire. It should at once be put into a wide-mouthed bottle containing a plentiful supply of the following solution:—

Bichromate of potass.	90 grains.
Glacial acetic acid	2¾ fluid dr.
Water	6½ ,, oz.

"In removing the brain of a rabid animal the greatest care must be taken not to allow the saliva or brain substance,, both of which, as we have seen, contain the rabies virus, to come in contact with any cuts or abrasions on the hands."

If the above instructions cannot be carried out in detail, the rougher and readier method of simply exposing the brain by the

removal of the anterior wall with a chisel and hammer, and the removal of a piece of the back part of the brain (medulla) under aseptic precautions, placing it immediately in pure glycerine and forwarding it to the laboratory, answers all practical purposes. It must be remembered that in most instances the brain will be considerably mutilated owing to the dog being unavoidably destroyed by shooting. In other cases the dog has perhaps had his brain battered in by sticks or stones, and may have been dead several hours before he is seen by a veterinary surgeon. In the latter case, as putrefaction in a tropical climate is so rapid, the brain substance is already infected, and hence the precaution of washing the skin before exposing the brain can hardly be considered necessary.

TREATMENT OF A PERSON BITTEN BY A DOG SUSPECTED OF RABIES.

In giving advice concerning the state of health of a dog which has bitten a human being, one is justified in erring on the side of safety, and if there is the slightest ground for suspicion that the dog in question, although at the moment of biting showing no pronounced clinical symptoms of rabies, might possibly be in the incubative stage of the disease, it is far better to exaggerate rather than minimize the possibility of infection to the person bitten. It must be remembered that there are many people who are not of a nervous disposition, and who will jump at any excuse rather than undertake a journey to a Pasteur Institute for antirabic treatment, the journey involving a good deal of discomfort and expense. Should any such person, owing to a hesitating opinion given by a veterinary officer, decide to take what might be a small risk and not undergo treatment, or delay making the journey until the suspected dog should show more definite symptoms—in either case with fatal results—the aforementioned veterinary officer would, under such circumstances, be very much to blame.

The wounds inflicted by any dog, whether suspected of rabies or not, should be immediately cauterized with pure carbolic or some other reliable caustic. In the case of a person bitten by a dog apparently in the best of health, and concerning which suspicions are not aroused, no further treatment is necessary provided the dog in question remains in good health for the succeeding ten days.

In the case of a person bitten by a dog to which the slightest suspicion can attach, that person should be advised to proceed immediately to the nearest Pasteur Institute for treatment. The dog meanwhile should be securely fastened and segregated and not destroyed. If the actual fact of its having bitten a person unprovoked is a clinical symptom of rabies, then within the following ten days the animal would develop unmistakable signs of the disease, in which case he should be destroyed and the brain forwarded to the Institute to which the person bitten has proceeded for verification. It must be remembered that the success of the treatment depends on the time which elapses between the moment of the infliction of the bite and the moment of commencing treatment. Hence there should be no delay.

If a dog which has bitten a person, the latter having proceeded to the nearest Institute for treatment, should be in the best of health ten days after having bitten such person, the fact should be communicated by wire to the Institute so that treatment can be suspended.

In the case of a dog which has been beaten to death after biting any human being or other animal, the brain should always be sent to the Institute if possible. This allows of the treatment of any person or persons being suspended should the case prove not to be one of rabies after experimental inoculation.

The Moment at which the Saliva of a Rabid Animal Becomes Infective.

Cases have been reported of infection resulting from the bite of a rabid dog many days before the dog actually showed clinical symptoms of the disease.

In 1890 Nocard and Roux showed that the mixed saliva is always virulent 24, and sometimes 48, hours before the appearance of any change in the appearance of the dog.

In 1901 Rabieaux and Guinard showed that siliva from the submaxillary gland, removed aseptically from an opening in Wharton's duct, might be virulent for two days before the appearance of any symptoms.

Nocard, Roux, and Nicolas noted that the appearance of fever indicated the secretion of virulent saliva.

The work of Nocard, Roux, Guinard, and Rabieaux and Nicolas show that the moment of infectivity of the saliva is variable.

The question, however, is a most important one, since it must be determined what is a safe period to allow between the moment of a dog, apparently healthy, biting a person and the moment of its showing clinical symptoms of rabies, before it is considered necessary for such person to undergo antirabic traetment.

To be licked on an abrasion must be looked upon as equally dangerous as a bite from the point of view of treatment.

Professor Galtier, of Lyons, always advised Pasteur's treatment being carried out on every person bitten within a period of eight days before the appearance of symptoms in the offending animal. This is certainly a safe period to adopt.

(*To be continued.*)

Clinical Articles.

OPHTHALMIA IN TETANUS.

By S. J. MOTTON, M.R.C.V.S.,

Penzance.

Changes in the eye occur in many diseases, but apart from the protrusion of the membrana nictitans, I have not seen anything recorded in connection with the eye in tetanus.

On October 10 of last year, an eight-year-old general purpose mare, was brought for inspection. The owner complained that for several days the mare had been moving stiffly.

The membrana nictitans covered almost the whole eye when her head was raised. Her jaws could be opened to only half the normal distance, the tail was elevated, and she walked with her hind legs widely separated.

Tetanus was diagnosed—the mare was placed in a box and treatment commenced. On October 16 she was much worse and was placed in slings. On October 19 (nine days after the first inspection) the cornea of the near eye was observed to be cloudy, and on the next day the off cornea was similarly affected. There was a thick, but not very abundant, discharge from both eyes, and in two or three days each cornea had assumed a pale yellow colour. The mare was now quite blind. In another week the opacity began to pass away.

On November 16, the animal was taken out of slings and made a good recovery. By the end of December the sight of both eyes was quite good. The right cornea was normal. The

left cornea was slightly blemished by a yellow speck about half the size of a B.B. shot.

Zinc-sulphate lotion was the only material applied to the eyes.

A CASE OF ABDOMINAL ABSCESSES IN A HORSE.

By Captain E. S. GILLETT, M.R.C.V.S.,

Army Veterinary Corps, India.

Subject.—Coarse-bred Australian gelding, four years old.

History.—Landed in India, January, 1910. He proved a very difficult horse to get into condition, although he carried a good coat and to all appearances was healthy.

In June he suffered from slight fever for ten days, but his temperature never exceeded 101·4° F. I had his weight recorded weekly from July 1, when he turned the scale at 980 lb., and he gradually increased to 1,068 lb. at the end of September; but a horse of his size should have weighed 100 lb. more than that. His temperature was taken daily during this period—July to end of September—and registered normal. I had him broken to harness and given slow work and special care, but he refused to thrive.

On October 5 I got a message in the early morning to say that he had been working quietly in a cart and had suddenly become unable to move; it was thought he had dislocated his shoulder. I arrived on the scene about half an hour after the occurrence, when the horse seemed all right, with the exception that he was soaked in perspiration, and I removed him to the Veterinary Hospital.

The line overseer reported to me when the horse was admitted that he had noticed this horse's "guts rumbled a lot," so much so that the native coachman who used to exercise him had called his attention to the noise on several occasions.

The horse after being admitted to hospital was carefully watched, though the reported increased peristalsis of his intestines was not noticed till three days before his death. His temperature from October 5 to 27 remained normal, pulse and respiration also normal, and he was simply kept under observation and carefully fed.

In spite of this he lost condition, and on October 27 his temperature rose to 102·6° F., and remained about 103° F. till he died on November 12 (chart attached). After a few days' per-

sistently raised temperature, and taking into consideration his condition, &c., I suspected one of three things (1) piroplasmosis; (2) internal abscess; (3) glanders (unlikely).

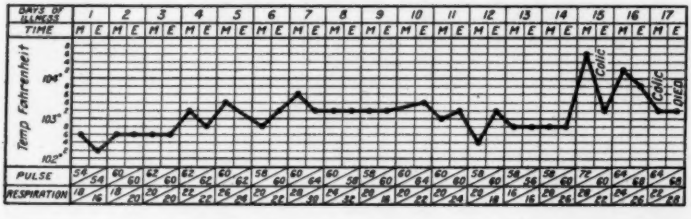
As regards (1) piroplasmosis, microscopical examination failed to show the presence of piroplasms, so I wrote for some mallein to settle the possibility of glanders, but did not receive it before the animal's death.

On November 10 the horse at 5 p.m. had an attack of colic, which yielded to ordinary treatment; marked peristalsis of the bowels was noticed.

On November 12, 6 p.m., colic recurred, accompanied by severe diarrhœa, and the horse died at 5 p.m. next morning.

Unfortunately I was away on purchasing duty the night he died, and also next day, so can only quote the Head Veterinary Assistant's report of *post-mortem* examination, which was as follows:—

Post-mortem Examination.—Large abscess joining stomach to spleen. Estimated to contain over one quart of pus. This abscess had burst into abdominal cavity; no peritonitis. Large



number of small abscesses on diaphragm; mesenteric edge of large colon contained three large abscesses. Internal lining of intestine normal. Liver weighed 25 lb.

Remarks.—This case appears to me to be unusual in several ways. The subject must have been affected for many months; at least I am of opinion his failure to thrive and come on was due to this, but until seventeen days before his death his temperature was normal. In other cases I have had, a persistent high temperature for weeks or even months has been the rule, and is, in my opinion, the chief aid to diagnosis of this condition. This horse would probably have survived some time longer if he had not had an attack of colic. The liver, which I saw myself,

weighed 25 lb., and was macroscopically healthy otherwise; in a horse of this size its normal weight would be about 11 lb. or 12 lb. The marked abnormal peristalsis of the bowels must have been connected with the disease, but I have never remarked it in other similar ones.

AN INDIAN METHOD OF RESTRAINT FOR CATTLE.

By Captain E. S. GILLETT, M.R.C.V.S.,
Army Veterinary Corps, India.

The attached photograph explains a very simple way of restraining cattle. Personally I had never seen it till I came to Southern India, but it may be, of course, known at home.

All that is required is two ordinary poles, which are crossed under the animal's belly in front of the hind legs, and easily held in position by two men.

It is quick, simple, and effective, and I have found it invaluable when inoculating large numbers of cattle against rinderpest, as even the wildest of cattle can be done without casting them, provided their heads are attached by a strong rope to any stable object.

THE USE OF ADRENALIN IN THE TREATMENT OF ACUTE LAMINITIS.

By Captain T. A. NICHOLAS, M.R.C.V.S.,
Army Veterinary Corps, Norwich.

During the recent army manœuvres there were two cases of acute laminitis among the horses of the 16th (The Queen's) Lancers.

Case 1.—A 47 was noticed at evening stables on August 30, 1910, to be off feed, dull. Temperature, 102°. Fore legs well forward and resting on heels, feet very hot, and throbbing of the digital vessels; groaning on being moved; quivering of muscles of fore limbs.

The hair was removed on each side of both fore pasterns (lower third), and skin painted over with iodized chloroform. Two tablets of adrenalin* (Parke, Davis and Co.) were dissolved in 2 dr. of boiled water. Thirty mimims were injected subcutaneously into each side of both fore pasterns, and cold bran poultices applied to the feet. Next day the horse, although still lame, was able to walk to the Field Veterinary Hospital (300 yards), and as far as I know no further injections were employed and the horse was discharged cured on September 12, 1910.

Case 2.—A 92 was discovered at evening stables on August 31, 1910, after a long march, to be off feed, dull, and showing symptoms of acute laminitis. Temperature, 102·4° F. The same treatment was adopted as in case No. 1, and horse was able to march well to camp on September 3, 1910, a distance of about 10 miles, and was discharged cured on September 4, 1910.

Note.—Both these horses marched back to Norwich from Salisbury Plain. Adrenalin should certainly be tried on all cases of acute laminitis.

* Each tablet contains 0·001 gramme adrenalin. One tablet dissolved in 1 c.c. of distilled water equals 1 000.

HERNIOTOMY IN A VALUABLE RUSSIAN HORSE.

By A. CHINNIAH, G.B.V.C.
Colombo, Ceylon.

History.—On the 20th May, 1909, I was informed by a telephone message that the horse "Lally," belonging to Mr. D. E. W. Pedris, the plumbago merchant, was suffering from an attack of colic. I proceeded to the spot at about two in the morning. As soon as I saw the animal I suspected the colic was due to hernia. I examined the animal per rectum, reduced the hernia and came away after suggesting to the owner that he was suffering from scrotal hernia and that he should be operated upon at no distant date. People of this country dread the word "operation" and I could not induce the man to have the operation done. After this I had an eye on the animal and made frequent enquiries as to his ailments, etc. He had several attacks of colic and they were treated accordingly, as per ordinary colic, by different people. Some time in December, 1909, the scrotum had assumed an enormous size and some practitioner suspecting the tumour to be due to hydrocele applied the strong tincture of iodine, and even the red iodide of mercury blister was put on. And as a result there was a certain amount of sloughing, leaving an ugly sore. Early in January, 1910, it was thrown down by a surgeon and operated upon. When the sore was opened, the gut protruded, and it was put back and stitched up immediately. In June, 1910, the owner brought the horse back to me and an operation was decided upon and performed successfully on the 3rd of July.

The delay till the 3rd of July was due to the want of hernia clam, which had to be made by Messrs. Walker, Sons & Co., Colombo, at my suggestion.

Preparation for the Operation.—From the 1st of July the horse was given bran mashes. On the second morning he was injected with a dose of antitetanic serum and in the evening too the dose was repeated.

Operation.—The instruments taken for the operation were:—scalpels, castration knife, sharp-pointed and blunt bistoury, director, Syme's artery forceps, curved needles, metal castration clam (made by me), and sharp and blunt scissors.

They were thoroughly boiled and sterilized for half an hour before the operation. The animal was thrown on the near side and general anæsthesia (chloroform) was administered. He consumed about eight ounces of chloroform and had to be kept under it for over two

hours. The penis was pulled out, washed and lubricated. The hernia was on the off side, and consequently, the off hind leg was liberated from the hobbles and held firmly by a strong syce. I took my usual position as in castration. The scrotum, as the result of continued meddling and futile blisters, was of a very large size and hence, unwieldy. The presence of excessive adhesions made me take great precautions, bit by bit was carved out carefully in a line parallel to the median raphe, instead of a bold, long, deep cut along it. This was rather a hard task. Till the tunica vaginalis was reached the structures had to be torn by the fingers. When the sac was reached a portion of the serous sac was carefully cut by the scissors, care being taken not to injure the hernia. There was a copious flow of serum. I did not use the director, but with the help of the finger I guided the bistoury in opening the sac and finally the gut was exposed. It being a very chronic case the adhesions gave me some amount of trouble. These being freed the gut was washed in a solution of boracic acid and returned into the abdomen. I have watched several cases of herniotomy in man, done by my friends at the General Hospital, and so I thought I should slit up the funicular portion of the scrotum as far as it was possible and put in a suture or two at the neck. There was some amount of bleeding, but the use of artery forceps had the desired effect. This slit in the scrotal sac enabled me to reach the inguinal ring with ease. With my two left fingers inside the abdomen I pulled one side of the ring up to the edge of the wound I made. Then the needle was passed from the right to the left and I was only able to put one suture with strong catgut. This done I thought I would try to preserve the ornaments of the poor beast, but I had to decide otherwise owing to the big slashing wounds due to the large extent of the adhesions. The slit along the funicular portion of the sac was sutured with continued sutures of strong silk cord.

Having decided not to adopt the conservative process of retaining the testicles, I then hurriedly opened the left sac, and felt the ring and found it free. The whole scrotum with the testicles were enclamped in my metal clam, and I screwed up the bolts very tight. I had to take a further precaution of putting a strong double suture, just above the clam, to prevent the descent of the gut in case the clam came off prematurely. Soon after washing the parts with mercury lotion the animal was on his fours none the worse for the long and tedious operation.

After Treatment.—In the Tropics, where the chances of quick

healing are less, I had to take a deal of trouble over the case. The animal was in a great agony for about four hours till the mortification of the enclamped scrotum and testicles had set in. I always adopt lotion treatment. A spray of carbolic lotion was kept on every now and then for several days. On the third day the scrotum and testicles were cut away about 3 in. below the clam. The clam dropped down on the eighth day, thus exposing a big cavity, which gradually filled up by granulation. The following dressing was applied :—

℞.	Acidi borici	℥i
	Iodoformi	ʒss
	Ol. eucalypti	
	Acidi carbolici	āā ℥ii
	Glycerini	ʒxii
	Misce.	

The animal was fit to be driven on August 8. He is now, I am pleased to say, in fine working order.

PRIMARY TUBERCULOSIS OF THE VULVA IN A COW.

By E. JQEST.

[*From the Report of the Veterinary College of Dresden for* 1909.]

The Pathological Institute received from Dr. Meyer in Weimar, the vulva with the posterior portion of the vagina of a slaughtered cow. The following history was sent: An 8-year old well-nourished cow had given birth to a calf five and a half months previously. Parturition had been effected by a quack who had caused a rent in the vagina. As a result of this injury thickening of the vulva gradually arose. At the *post-mortem*, besides the vulval disease the cow only showed tuberculosis of the bronchial lymphatic glands, the remaining organs, especially the newly-examined uterus, were quite free from tuberculous changes.

Pathological examination showed great thickening of the vulval lips, especially in their transverse diameter. They were swollen to the size of a man's head and their pressure had caused a prolapse of the uterus. The weight of the vulva with the posterior portion of the vagina was 2½ kilogrammes (5¾ lb.). The consistency was hard. The diameter of the right lip was 8 cm. and of the left 6 cm. The clitoris was pushed out of position and was 8 cm. in front of the

ventral angle of the vulva. The cut surface of the thickened vulval lips was greyish yellow in colour, there were caseo-purulent masses in it, and greyish-white connective tissue strands. The tuberculous masses were between the muscle and connective tissue bundles.

Bacteriological examination showed tubercle bacilli. Experimental animals (guinea-pigs) showed tuberculous illness after two to three

weeks, and were killed at the end of six weeks and found to be badly affected with generalized tuberculosis.

In this case the tuberculosis was primary, for on *post-mortem* examination, except for the bronchial glands, the cow showed no internal changes and the uterus especially was normal. The infection probably occurred through the injury occasioned five and a half months previously.

Canine Clinical Notes.

THE TREATMENT OF ECZEMA BY INTRAMUSCULAR INJECTIONS OF A SOLUTION OF COLLARGOL.

By J. A. N. DA CUNHA, G.B.V.C.,
Veterinary Officer to the Government of H.H. the Sultan of Zanzibar.

Subject.—Terrier dog "Slippers," the pet of an army officer's wife.

Symptoms.—Small, irregular eruptic patches excessively sore and tender, covered with encrustations and matting of hairs scattered about the neck, shoulders, dorsal column, and hind quarters, and a few pustules varying in size from a millet seed to a pea under the elbows, belly and inner surface of hind limbs. Great irritability of the skin and intolerable itching, which caused the animal to rub itself continuously against the ground.

Treatment.—All hair was shaved off, the irritable spots cleaned with soap and warm water, and when these were dried different dressings were well rubbed in to allay itching. The eczematous areas were anointed with boric acid and zinc-oxide ointment. The cause of the disease being dietetic in origin, measures were taken to rectify it by administration of purgatives, general tonics, careful diet and exercise.

A large number of well-known remedies, both internal and external, were severally tried for nearly a month and a half, but every endeavour to cure this disease failed.

Fresh eruptions appeared every day over the neighbouring parts, the usual scratching continued and the disease assumed a form of a very annoying character, with strong tendency to recurrence.

The writer's attention was attracted to the method of treating eczema in dogs by collargol, as recorded by Mr. E. Pignet in a veterinary periodical, and his line of treatment was tried as follows:—

Intramuscular injections of 3 c.c. of 1 per cent. solution of collargol every alternate day into the muscles of the thighs, and an external application of the same solution to the sores. On the ninth day the itching irritation subsided, and in another ten days every vestige of this troublesome disease had nearly gone. A slight lameness ensued after each injection, but in every case it disappeared in twenty-four hours.

Subsequently the writer had the opportunity of trying this drug on another similar case of acute moist eczema, with equally successful results and in a shorter space of time.

Argentum colloidale—Colloidal silver—Collargol "is prepared by mixing sulphate of iron and silver nitrate. It occurs in the form of small black, metallic-looking grains, odourless, soluble in water, 1-25 forming a brownish-black solution, and has no caustic action."

The action of collargol is not antibacterial but antitoxic, and this action is supposed to be due to artificial catalyses. It is chiefly used in the form of intravenous injections or intramuscular in the case of small animals, in the doses of 2 to 5 c.c.

THE USE OF ASPIRIN IN CANINE PRACTICE.

By Captain E. S. GILLETT, M.R.C.V.S.,
Army Veterinary Corps, India.

I have lately tried aspirin in cases of dogs suffering from persistently high temperature with exceedingly satisfactory results. The first case I used it on was a pedigree fox-terrier puppy of my own out of imported parents. He had a temperature of 106° F. for a week, often going over 107° F., and refusing all food.

I tried all the ordinary febrifuges, &c., but without the least effect, and eventually administered aspirin in tabloid form. The effect was marvellous—temperature was at once lowered, and in three days the pup was normal, and eating Benger's food greedily and never looked back again.

I have been equally successful in three or four other cases, and think it is well worth a trial by those who may not have already used it.

The depressant effect so well marked after the exhibition of phenacetin and antipyrin is absent.

Abstracts and Reports.

MILK SUPPLY.[1]

Instructions for ensuring the supply of Clean Milk.

To Farmers and other Milk Producers.

The Cows.—The cows should be healthy, and to that end they should be kept in the open air as much as possible. Farmers are advised in their own interest, as well as in that of the public, to buy only cows about the health of which they have no doubt, and to keep cows that may happen to fall ill on their farm isolated from other cows.

The cows must be kept clean. For this purpose plenty of good bedding must be provided and regularly renewed. The stalls and manure-channels must be properly constructed.

The udders and teats of the cows must be cleaned with a clean damp cloth before milking. If the udder is soiled so that it cannot be cleaned in the way mentioned, it should be washed with soap and warm water and carefully dried with a clean cloth.

The milkers should be instructed to examine the cow's udders and teats at every milking to ascertain if there is any unusual lump or swelling present. The best time to make the examination is immediately after milking, when the udder is empty. If anything suspicious is found, the advice of a veterinary surgeon should be obtained at once. When there is obvious disease of the udder or teats, the milk should not be sold or used for human food, until a veterinary surgeon has made an examination and decided whether it is safe so to use it.

The Milker.—The milker must wash his or her hands, using a nail-brush, before milking. Soap, towels, and water must be provided in a convenient place for this purpose. A clean linen smock should be worn while milking; and then only. Persons who are poorly or have sickness at home should have nothing to do with the cows without the sanction of a medical man.

The Cow-shed.—The cow-shed should be so lighted that every part of it is easily visible in the daytime with the doors closed.

The cow-shed should be so ventilated that the air in it will not feel close, or have a disagreeable smell, when the cows are all housed and the doors shut. Cows, like human beings, will not suffer from ample ventilation if it is steadily maintained throughout the whole year.

The manure-pit should be so situated as not to affect the fresh-air supply of the cow-shed, and should be as far as possible from any place where milk is kept or dealt with.

Horses, swine, poultry, or pigeons must not be kept in the cow-shed, or in any loft over it, or in any building communicating with it.

[1] Leaflet A prepared by the Joint Committee on Milk of the National Health Society and the National League for Physical Education and Improvement; and published by the League at 4, Tavistock Square, W.C.

Leaflet B contains instructions to Distributors and Retailers of Milk and Leaflet C to Housewives and all Consumers of Milk.

Price: 6d. per doz.; 2s. per hundred; 17s. 6d. per thousand.

The cow-shed must be kept as clean as possible, and have an impervious floor. The ceilings and under surface of the roofs should be cleared of dust and cobwebs at least every three months, and the walls and ceilings lime-washed every six months, and more frequently if necessary. The manure should be taken out not less frequently than twice a day, and the manure-channel and walks should be flushed with water daily at least.

Hay or other food must not be stored in the cow-shed. If hay or other food is stored in a loft over the cow-shed, the trap-doors or other communications with the cow-shed must be kept closed except during the feeding or foddering of the cows.

Every effort should be made to keep the cowshed free from dust, especially at milking times. Foddering or sweeping which creates dust should not be done just before or during milking time.

The milk should not be left standing in the cow-shed, but immediately after each cow is milked her milk should be removed to a suitable place used solely for milk purposes, free from dust or other sources of contamination, and at once strained and cooled.

Utensils.—All milk vessels should be so constructed that all parts can be thoroughly cleaned; they should be without recesses that are difficult to clean.

Milk cans should be so constructed that every part inside is easily cleaned; and when the lid is removed no dust or water falls into the can. Lids must be close-fitting and dust-proof; they should be sealed or locked in transit.

Every milk vessel must have a mouth wide enough to admit the hand, and should after each use be thoroughly cleaned with a wet cloth and rinsed out, and finally scalded with boiling water.

All cloths used for cleaning milk vessels should be frequently wrung out of clean water and should be boiled once daily.

The milk utensils should not be allowed to come into contact with any source of contamination, either when being cleansed or at any other time, and must only be used for milk. For example, milking pails must not be used for watering the cows, and milk utensils must not be washed along with clothes.

It is necessary to have separate accommodation for washing milk utensils, and a separate place for storing them.

An efficient sieve must be provided for straining the milk, and must be maintained in good condition. Muslin or other suitable material must be used with the sieve. The muslin should be washed and scalded immediately after use.

An efficient refrigerator or other means of cooling milk is recommended, as cooling is the best means of keeping milk. The cooling should be carried out in a place which is free from dust or other sources of contamination, and the water used in the refrigerator should be pure.

The refrigerator and the sieve must be treated in a manner similar to the milk vessels, and kept thoroughly clean.

Nothing should be added to the milk for preservative or any other purpose.

Storage.—The place where the milk is stored or sold should be used for no other purpose. It should not communicate directly with the living rooms, and should be adequately ventilated and lighted.

Ventilating openings should be guarded with gauze metal screens to prevent entrance of flies. The milk should be distributed with as little delay as possible.

Human Infection.—Every effort should be made to keep the operations in connection with the milk business separate from household work, so that if infectious disease attacks the family the infection may be kept away from the milk. As children may suffer from unrecognized forms of infectious disease, no young children should be allowed to enter the place where the milk is stored, or take any part in handling the milk or milk utensils.

To be hung up in every Cow-shed.

CHIEF SOURCES OF CONTAMINATION OF MILK AND MEANS OF AVOIDING THEM.

SOURCE OF CONTAMINATION.	REMEDY.
Cow—Dirty udder	Wash udder before milking.
Diseased udder	Do not use the milk. Send for veterinary surgeon.
Milker—Clothes...	Wear clean overalls.
Hands	Wash hands before milking.
Illness	Those who are poorly, or who have sickness at home should cease milking until sanctioned by medical attendant.
Cowshed—Air and dust	Avoid foddering and sweeping at and before milking. Remove milk from cow-shed as soon as possible.
Utensils—Milk-pails, Cans and Coolers.	Cleanse thoroughly by steam or boiling water.
Storage—...	Store only in a clean, cool, and airy place, protected from flies and dust.

A METHOD FOR ISOLATING AND GROWING THE LEPRA BACILLUS OF MAN AND THE BACILLUS OF JOHNE'S DISEASE IN CATTLE. (PRELIMINARY NOTE.)

Indexed B. A. I.

BY F. W. TWORT, M.D.,
The Brown Institution, University of London.

FOR a number of years different investigators have attempted to cultivate the lepra bacillus of man and the allied organisms found in the rat and other animals. It is not intended in this preliminary note to discuss the numerous papers which have been published from time to time from the various English, Continental, and American laboratories. These papers deal with non acid-fast bacilli, or with acid-fast bacilli growing quickly on ordinary media, which, in the opinion of the writer, are contami-

nating organisms, and not the true lepra bacillus. So far, no one has produced a culture of acid-fast bacilli isolated from a leper, and showing the characters of the lepra bacillus as found in the tissues of man. It was with the object of obtaining a pure living culture of the lepra bacillus that these investigations were undertaken.

The material used was the nasal discharge and scrapings from a typical leper. The discharge showed large masses of lepra bacilli and a number of contaminating micro-organisms. Firstly, most of the contaminations were killed by placing the discharge in a 2 per cent. solution of ericolin at 38° C. for one hour as recommended for the isolation of the tubercle bacillus*; then cultures were made from the sediment on to different media and incubated at 38° C.†

All the ordinary laboratory media, including Dorset's egg medium, gave negative results. A number of special media containing extracts of fresh gland and other tissues were tested next, the extracts being freed from any contaminating micro-organisms by passing them through a Doulton white filter; these also gave negative results.

In view of the close relationship between the tubercle bacillus and the lepra bacillus, it appeared highly probably that these two organisms would require the same chemical substances for building up their protoplasm, which could be elaborated from the ordinary media only by the tubercle bacillus. It was thought that if these substances could be supplied, already formed, to the lepra bacillus, it might grow, and the easiest method of supplying these substances would be by adding to some good medium the ground-up bodies of tubercle bacilli containing them. Accordingly, a number of tubercle cultures were taken and inoculated on to Dorset's egg medium; when sufficiently grown the tubes were steamed and the growth of tubercle scraped off the surface, care being taken to avoid the medium containing the waste products of the tubercle growth. The tubercle bacilli so obtained were ground up with glycerine and saline, steamed for half an hour and added to the yolk and white of new-laid eggs in the following proportions:—

Eggs, 75 parts; 8 per cent. sodium chloride, 25 parts; mix well and add tubercle bacilli, 1 per cent.; and glycerine, 5 per cent., or less.

The medium was placed in test-tubes, heated to 60° C. for one hour, and on the following morning incubated at 38° C. for six hours, and again heated in water bath at 60° C. for one hour, and set in slopes at 85° C.

The ericolinized nasal discharge of a leper was inoculated on to this medium,‡ the inoculated tubes being capped with gutta-

* Twort, *Proc. Roy. Soc.*, B., 1909, vol. 81.

† Subsequent experiments have shown that 37° is a better temperature (November 19, 1910).

‡ After inoculating the medium with lepra material fresh human blood should be pipetted over the surface.—F.W.T.

percha tissue and incubated at 38° C. After 24 hours the medium absorbed a quantity of the ericolin, so the material was lifted off with a platinum loop and rubbed over fresh tubes. The bacilli grew and were sub-cultured in pure growth, the bacilli growing in sub-cultures as fairly long thin beaded rods; the bacilli were well formed and quite acid-fast. The lepra bacillus inoculated on to this medium at first grows extremely slowly, but later growth becomes faster, marked microscopic evidence being obtained in about four weeks. To the naked eye, growth is only just visible after about six weeks, appearing as a colourless film along the needle track. Attempts to sub-culture on to ordinary laboratory media are always negative.

Experiments are now being carried out, using other organisms than the tubercle bacillus for making the medium, and also testing various micro-organisms on the medium.

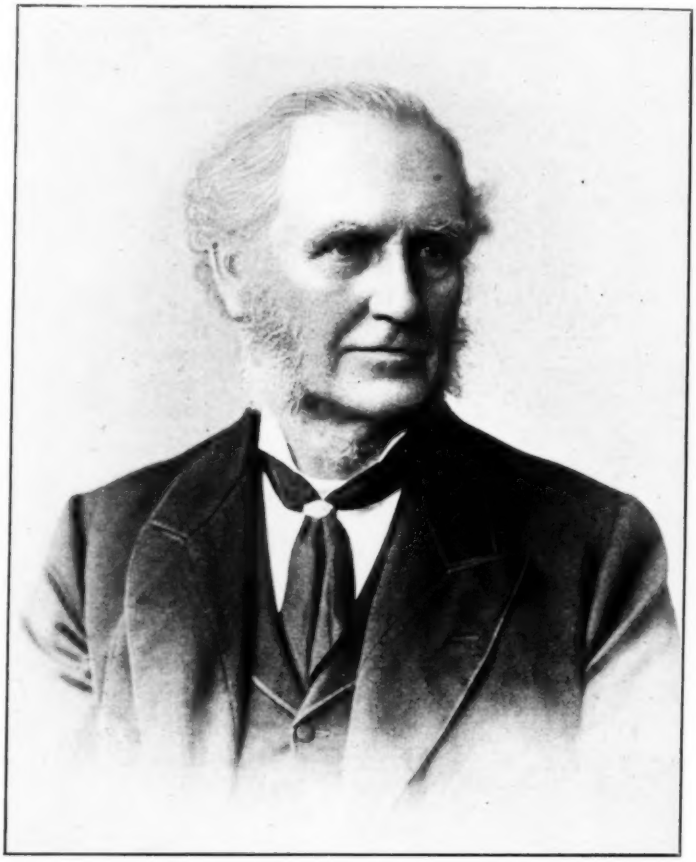
In the near future it is hoped to prepare a vaccine from the ground-up lepra bacilli, for the treatment of man suffering from leprosy. An attempt to grow the rat lepra bacillus on the same medium will also be made if the material can be obtained.

In conclusion, I may note that, working with Mr. Ingram, M.R.C.V.S., on the same medium I have also succeeded in isolating and growing the acid-fast bacillus found in the intestine of cows in Jöhne's disease. The first generation of this bacillus grows often long, with occasional branching and club formation, in sub-cultures it gradually grows smaller, and in the second or third generation is about the size of the tubercle bacillus. The growth is only just visible to the naked eye, and sub-cultures on the ordinary laboratory media show no evidence of multiplication. Jöhne's bacillus grows somewhat more easily than Hansen's lepra bacillus, the bacilli being well formed and quite acid-fast. The cultures were incubated at 40° C. It is hoped, when sufficient material is obtained, to prepare a vaccine for diagnostic purposes.

Further details of these experiments will be published later.

(*Proceedings of the Royal Society*, B., vol. 83.)

N0562 p. 156 dec. 19. 1910



DR. HENRY POWER.

Late Professor of Physiology at the Royal Veterinary College, London.

HENRY POWER, 1829—1911.

Born at Nantes, in France, September 3, 1829, only child of Captain John Francis Power, 35th Regiment, by his second wife, Hannah, daughter of Henry Simpson, of Meadowfield, Whitby, Yorkshire. Immediately after his birth Henry Power was taken to Barbados, where his father was quartered, and narrowly escaped death in the great hurricane which devastated the island on August 11, 1831. Major Power resigned his commission in 1833, and for many years spent a wandering life, during which his son was sent to many different schools, amongst others to Cheltenham College. He was apprenticed to Mr. Thomas Lowe Wheeler in 1844, and entered at St. Bartholomew's Hospital. Botany attracted him especially, and he was awarded the Linnæan and the Galen medals at the Society of Apothecaries. In 1851 he was admitted a Licentiate of the Society of Apothecaries and a Member of the Royal College of Surgeons of England, and on December 1, 1854, he was elected a Fellow of the Royal College of Surgeons. At the University of London he had a brilliant career. He took the prize for chemistry at the matriculation; the exhibition and gold medal in anatomy and physiology and the gold medal in structural and physiological botany at the intermediate examination in medicine in 1852; the scholarship and gold medal in physiology and comparative anatomy, and the scholarship and gold medal in surgery at the final M.B. examination, graduating M.B. in 1855.

In 1851 Mr. Power was appointed Demonstrator of Anatomy at the Westminster Hospital, where he was afterwards appointed assistant surgeon. In 1855 he became assistant surgeon at the Royal Westminster Ophthalmic Hospital, and ten years later he was elected ophthalmic surgeon at St. George's Hospital, a position he resigned in 1870, when the department for diseases of the eye was instituted at St. Bartholomew's Hospital, and he was chosen the first ophthalmic surgeon.

He filled the office of Professor of Physiology at the Royal Veterinary College from 1881-1904, and on his retirement he was much touched by the presentation from the members of his class of an arm-chair fitted with an electric reading lamp and furnished with an appropriate inscription, as well as by a separate presentation from veterinary surgeons—former pupils—in South Africa who had been too late in contributing to the original testimonial.

At the Royal College of Surgeons of England he filled nearly all the official positions. He was a member of the Board of Examiners in Anatomy and Physiology from 1875-1884; and in physiology only from 1884-1886, when his place was taken by his eldest son, Mr. D'Arcy Power. He served two terms of office as a member of the Council from 1879-1890, and was a vice-president for the year 1885. He delivered the Arris and Gale lectures on anatomy and physiology, 1882-83; was Hunterian Professor of Surgery and Pathology, 1885-1887; Bradshaw Lecturer in 1886, and Hunterian Orator in 1889. At the University of Oxford he was an examiner in the School of Natural Science; at Cambridge

he was an examiner in the Natural Science Tripos; he examined at the University of Durham, and at the University of London he was an examiner in Physiology.

He served the office of president of the Harveian Society of London for a double term of office, 1880-81. He was president of the Ophthalmological Society of the United Kingdom, 1890-93, and was a vice-president of the Royal Medical and Chirurgical Society, 1892-93. For many years he was surgeon to the Linen and Woollen Drapers' Benevolent Fund and to the Artists' Fund.

He married his first cousin, Ann, daughter of Thomas Simpson, Esq., of Meadowfield, Whitby, Yorkshire, on December 21, 1854, by whom he had a numerous family, of whom four sons and four daughters survive.

The death of Sir William Savory, his lifelong friend and companion, in 1895; of Mr. Bowater J. Vernon, his colleague in the eye wards, with whom he was in daily communication in 1901; and finally the tragedy which drowned his artist daughter and his granddaughter before his eyes at Whitby, when he was himself rescued with difficulty, so saddened him that he decided to leave London for a less exacting life in more congenial surroundings.

Mr. Power was a skilful artist both in watercolours and with a brush and Indian ink, and in early life he played the violin.

He was interred at Whitby on Saturday, January 21, amid many tokens of respect from his fellow-townsmen and with a wealth of flowers sent from all parts by his numerous friends.

He wrote (1) "Illustrations of the Principal Diseases of the Eye," the original watercolour drawings being made by himself from patients who came under his care at the Royal Westminster Ophthalmic Hospital. (2) "A Text-book of Human Physiology" (Messrs. Cassell). (3) "Articles for Professor Axe's Monograph on the Horse.". (4) "The New Sydenham Society's Lexicon of Medical Terms" (with Dr. Leonard W. Sedgewick). (5) "Translations from the German of Stricker's Manual of Human and Comparative Histology," in 3 vols.; "Kramer on the Aural Surgery of the Present Day"; "Erb on Diseases of the Peripheral Cerebrospinal Nerves."

Reviews.

SPEZIELLE PATHOLOGIE UND THERAPIE DER HAUSTIERE. By Hutyra and Marek. (Special Pathology and Therapy of the Domesticated Animals.)

IT is hardly two years ago since a second edition of this work was published and reviewed in the pages of this journal. A third edition is now on the market, showing that the volumes have been eagerly taken up and used by the reading professional public. In order to keep pace with advancing scientific knowledge in connection with the conferring of immunity and diagnosis of disease by means of sera reactions, a third edition was necessary, and these new methods of investigation obtain full consideration in the present volumes.

Tuberculosis, glanders, contagious abortion and swine fever all receive amplified review. Tropical illnesses also have more extended notice. Fresh chapters have been added on the leucæmia of hens, dochmiasis or "salt sickness" in cattle (Southern Texas, Florida, and Bohemia), enzootic cretinism in animals, serum illness and necrobacillosis. Up-to-date information on Jöhne's disease is given, but it is questionable whether English investigators will agree with the names "spezifische paratuberkulöse Därmentzundung der Rinder" or "enteritis paratuberculosis bovis specifica" used. It is rather strange and surely an error—or is it a posthumous joke?—that the only synonym for the disease, viz., "Jöhne's disease," is given as an English one, and an English one only. We fancy that the disease would be known and recognized by this name in many other countries besides England, seeing that it has been noticed in Germany, Denmark, Holland, France, Norway, Switzerland, Hungary, and North America. There is an excellent illustration of the disease.

Descriptions are given of the palisade and red worms which were recently the subject of some controversy.

Our most up-to-date investigators will not consider the nomenclature as satisfactory. The names given are: (1) *Sclerostonum equinum*; (2) *Sclerostonum vulgare*; (3) *Sclerostonum edentatum*; (4)*Cyathostonum tetracanthum*. Perhaps of more importance than the nomenclature is the treatment adopted against these worms on the Continent, which consists of subcutaneous and intravenous injections of atoxyl (natrium arsenicosum) in gradually ascending doses. Intensive nourishment of the suffering subjects is also advised.

Hodgkins' disease is described as a pseudoleukæmia, but the picture of the dog given on page 870 as suffering from lymphatic leukæmia looks very like a clinical illustration of Hodgkins' disease. Several good coloured plates have been added to the work, notably those of contagious vaginitis, the ophthalmo reactions in glanders and tuberculosis, and pustulous acariasis in the dog. The chapters on skin diseases are a strong feature of the book and well worthy of the perusal of all veterinarians; even a glance at the illustrations being educative if one cannot understand the text.

Notwithstanding the additions and alterations in the volumes the compass of the tomes is not greatly increased. The spelling of the word "rachitis" is still original, and with close proof reading ought to have been corrected.

Nothing but praise can be given to the authors of the book, and their industry and facilities for acquiring thorough information must have been great. Finally it may be written that the book is destined to last and be of great and valuable service to veterinary surgeons and medical men; it is published in two volumes by the firm of Gustav Fischer, of Jena; the literature overhauled for the compilation of the text dates, with few exceptions, right up to the end of May, 1910. The work has already been translated into Italian, and Russian editions are in process of publication.—G. M.

THE JOURNAL OF MEAT AND MILK HYGIENE. Bale, Sons and Danielsson, Ltd., London. No. 1, Vol. I., January, 1911, pp. 64. 1s. net post free.

WE have received from the publishers a copy of a new public health journal, which should prove of interest to veterinary surgeons occupied in the practice of Municipal or State Veterinary Medicine. If succeeding numbers come up to the standard of number one there can be little doubt but that the journal has come to stay. We notice among the contents of the issue before us an article on "Meat Poisoning" from the pen of Professor E. J. McWeeney, Examiner to the Royal College; an article by Dr. Hume Patterson, Bacteriologist of the County of Lanark, and a contribution from Dr. Dittmar, the Scottish Local Government Board Medical Inspector, on "The Public Slaughterhouse System of Scotland." Editorially the Tuberculosis Order is discussed, also the subject of a Uniform System of Meat Inspection. Veterinary contributions also appear, and these are from the pens of Mr. A. M. Trotter, of Glasgow, and Mr. Thomas Parker, of Newcastle-on-Tyne. One more article should be mentioned, though it by no means completes the list, and that is an article on "Edinburgh Slaughterhouses," which is illustrated by photographs and a plan. The number of veterinarians employed in public health work is small, but we heartily recommend them to invest a shilling on our new contemporary, and we sincerely wish it every success in bringing about its avowed object of securing for Great Britain and Ireland a satisfactory meat and milk supply.

MERCK'S INDEX. By E. Merck, of Darmstadt. 392 pages, 6s. 6d., post free, from the London office of the firm in Jewry Street.

THIS work contains a short *résumé* of most of the medicaments at present used in the medical and veterinary worlds. Arranged in alphabetical order, reference to any therapeutical agent is quickly effected. German, French, English, Italian, &c., preparations figure in the list and make the index quite cosmopolitan in character. The healing agents are divided into three chief parts, comprising (1) preparations; (2) drugs; and (3) minerals. In the drug list we get the etymology, source, family,

common name, country of origin, constituents, and last, but not least, the chief uses of any agent under consideration. The index is very complete and contains up-to-date information in a concise form of almost any and every medicinal article employed by science in combating disease.

Animal derivatives receive ample consideration, and we notice acceptable information about such recent introductions into pharmacy as yohimbine, peroxide of hydrogen, suprarenin, nuclein, chloretone, &c. The *veterinary uses* of any agent receive special notice. The book may be well described as up to date, concise, and comprehensive, and the matter contained in it indicates a very large amount of research on the part of its author. The work has large pages and is well bound, both of which are points in its favour.

Translations.

A NEW OPINION ON THE ETIOLOGY OF MILK FEVER.

By KREUTZER.

None of the causative factors up to to-day is sufficient. The *theory of intoxication* neither explains the good effects of intra-mammary injections nor the absence of the illness in cows in a poor state. It does not enable us to understand why disinfection of the intestine and repeated milkings are without favourable action on the course of the malady. The *theory of infection* has against it this fact, that the microbes described as pathogenic are normal hosts of the teat or genital passages, and that they have never been able to reproduce milk fever experimentally. Besides, how can one understand the absence of fever and the good effect of insufflation of air?

One can also raise numerous objections against the *theory of circulatory trouble* which puts the symptoms observed to the account of a cerebral anæmia following afflux of blood to the udder. Why are the symptoms produced generally two days after calving? Why are they sometimes observed before calving? Why does the paralysis spread slowly to the posterior limbs, then to the anterior and then to the neck? Why are bad milkers exempt? Why are medicines that modify the circulation without action?

According to the author, everything may be explained when one considers the circulatory disturbance of the lymphatic system and when one seeks in the lymphatic system of the udder the point of departure of the malady. At the moment when lactation establishes itself, the lymphatic circulation becomes very active and a large quantity of lymph is diverted into the afferent vessels, into the sub-lumbar glands, and gradually into the cistern of Pecquet. This rapid flow of lymph provokes an increase of pressure which makes itself felt as it mounts in all the affluent lymphatics and provokes at the level of their roots of origin modifications in the conformity and conductibility of the nerves. Thus is explained the successive appearance of

digestive troubles by modifications of the nervous terminations of the sympathetic and of the gastric and mesenteric plexus, of paralysis of the posterior members (lymphatic stasis in the posterior marrow), paralysis of the anterior members, then of the neck, and finally brain symptoms; "naturally a weak pressure only suffices to provoke functional disturbance of the brain and cord." Weakness of the heart will be a result of paralysis of the vagus. The absence of inflammatory reaction, the progressive paralysis will explain the lowness of the temperature. The snoring noise will be due to compression of the recurrent by the lymphatic trunks of the neck. Finally, this hypothesis enables us to understand the origin of serous collections found at the autopsy in fatal cases (lymphatic stasis). If the cow is the only domesticated female affected with milk fever it is because the has an udder much more functionally active than that of others and it is precisely in the best milkers that the malady occurs; rapid and abundant secretion accompany each other; in fact, a sudden and intense lymphatic flux which causes the symptoms observed. The frequency of the illness after the third calving is because in the cow lactation attains its maximum at this time. The evolution of mllk fever before or a long time after calving is caused by sudden variations of the secretion; indigestion, for example, leads to a great diminution of milk, followed by an increase when cure occurs. If the injection of air under strong pressure effects immediate cure it is because it suspends all lymphatic circulation in the udder which permits the general circulation to take up its normal course.

(*Münchener tierärztliche Wochenschrift, ex Revue Générale de Médecine Vétérinaire.*)

DOUBLE-SIDED INFLAMMATION OF THE LUNGS IN A HORSE THROUGH BREATHING SULPHUROUS ACID VAPOUR.

By Dr. HEUSZ.

Staff Veterinary Surgeon.

The rare occurrence of poisoning through breathing sulphurous anhydride in domestic animals seems to justify the recording of the following case. It occurred in the practice of Herr Müther, abattoir director, and I was called in in consultation.

The stall in which the horse was stabled was situated quite close to a cooling establishment in the Pictet system. Contrary to order the fluid in an enclosed vessel was allowed to flow out on the ground. The liberation of the gas in the neighbourhood had the tragi-comic effect of causing violent coughing among the ladies inhabiting a two-storied house close by, the fumes coming up into the house and necessitating hasty and thorough aeration of the rooms. Beyond an airing of the stable near the cooler, nothing was done and nothing suspected. In the morning—about twelve hours after the accident—when the horse was to be fed, it was noticed that the stable was full of a stinking smell. The animal was immediately let out, and on account of need of air and general unrest it was brought under veterinary treatment.

The animal was a brown mare, aged 11, of Belgian strain. Visible mucous membranes reddened. Pulse 60 and easily perceptible. Temperature 40·5° C. Pumping breathing with distended nostrils, respirations 35. On auscultation of the lungs on the under and middle third there was a rattling noise in union with whistling. In the upper third of the right lung sharp vesicular breathing was noticeable. The appetite was gone. The animal was brought into a roomy, airy stall. The thorax was wrapped in blankets wrung out of hot water, and the patient received subcutaneous injections of camphorated oil, and internally electuary of acetate of soda.

On the second day the patient stood apathetically and could only be moved with difficulty. The pulse was imperceptible. On the mucous membrane of the nose there were dark red, sharply defined spots about the size of a 10-pfennig piece. From both nostrils there was a discharge of slimy fluid mixed with blood. Breathing laboured and about 46 per minute. There was severe spontaneous coughing appearing whenever the animal made the slightest movement. Only a little hay was taken, and dung was frequently voided in small balls covered with slime.

On the third and fourth day the position was much the same. The conjunctival membrane showed a watery appearance with a yellow streak.

On the fifth day, temperature 39·8° C., respiration 32. Examination of the urine revealed it to be yellowish brown, turbid and opaque and full of threads. Reaction strongly alkaline. Sp. gr. 1036 and traces of albumin and much mucin.

On the sixth day the state of the lungs showed slight improvement, there being vesicular breathing in most of the right lung area. General condition much the same, but the horse had lain down for the first time.

On the seventh day visible improvement, hay and oats eaten. Nasal discharge more copious, greyish-white and slimy. Pulse 72, breathing 24, with pronounced flank movement.

On the eighth day further progress.

On the ninth day lung sounds almost normal. Cough less and breathing performed without special trouble.

On the eleventh day the horse was put to light work. Ten days later it had only a slight cough and quick breathing when exerted much. These symptoms disappeared a few weeks later.

(*Zeitschrift für Veterinärkunde.*)

THE INSPECTOR'S NOTE-BOOK.

Abridged Technique.

Hare or Rabbit?—A domestic rabbit is exposed for sale in its skin and not eviscerated with a view of making it pass for a hare. How can one distinguish one from the other? Look at the extremity of the ear, which has a black pouch or pocket in the hare, even in the white hare. This black pocket does not exist in the ear of the rabbit.

Young Rabbit or Warrener?—A domestic rabbit exposed for sale in its skin and not eviscerated will be distinguished from a wild rabbit by

the length of its ears. Take the ears and lay them down forwards; if they do not pass the end of the nose, it is a wild rabbit; if they are longer, it is a young domestic rabbit.

Roebuck or Deer?—Important diagnosis, for the deer may be sold as roebuck; the latter is more valuable than the former. The deer has an eye vein and possesses a long tail, very apparent. The roebuck has no eye vein; its tail is hardly visible.

Head of the Domestic Duck or Head of the Domestic Goose?—Easily differentiated by the following: The nostrils of the duck are situated at the base of the superior mandible; the nostrils of the goose are discernible at an equal distance from the base and the extremity of the mandible. If one pulls away the superior from the inferior mandible, it will be seen that the buccal roof of the duck forms a vault, and that of the goose is flat, with asperities.

In what State of Putrefaction ought we to reject Game which is "very high"?—When the hair or the feathers can be easily pulled out, and when by palpation there is crepitation, denoting putrid emphysema in the muscular tissue.—*L'Hygiène de la Viande et du Lait.*

Letters and Communications, &c.

Captain Gillett; Captain Nicholas; Mr. E. Wallis Hoare; Professor W. L. Williams; Professor Gofton; Professor J. T. Share Jones; Dr. Twort; Dr. Bagshaw; Mr. S. J. Motton; Mr. A. Chinniah; Mr. J. A. N. da Cunha; The Director General Army Veterinary Service, Mr. H. A. Reid; Mr. Jowett; Mr. Bevan; Mr. D'Arcy Power; The Anti-vivisection Society.

Books and Periodicals, &c., Received.

Journal of the Royal Army Medical Corps; Bulletin of the Bureau of Sleeping Sickness; The Representative Board of Agriculture and Fisheries; Department of Agriculture and Technical Instruction for Ireland; Ecyclopædia of Sport (Mr. Heinemann); Agricultural Journal of the Cape of Good Hope; Merck's Index; The Journal of Meat and Milk Hygiene; The Animals' Friend.

NOTE.—All communications should be addressed to 8, Henrietta Street, Covent Garden, London, W.C. Telephone, 4646 Gerrard. Telegrams, "Baillière, London."

Letters for the JOURNAL, literary contributions, reports, notices, books for review, exchanges, new instruments or materials, and all matter for publication (except advertisements) should be addressed to the Editors.

Manuscript—preferably type-written—should be on one side only of paper, marked with full name of author.

Illustrations for reproduction should be in good black or dark brown on white paper or card.

Advertisements and all business matters relating to the JOURNAL should be addressed to the publishers, Messrs. Baillière, Tindall and Cox.